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■ Stress Fractures

From:

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Editor:

We read with interest the article by Drs Anderson and Greenspan in the April 1996 issue of *Radiology* (1). In this article, the authors adopt the concept that stress fractures consist of two types: fatigue fractures and insufficiency fractures. This concept is widely accepted (2-4). In the same article, however, the authors state that "Unless otherwise denoted, the term 'stress fracture' will refer to this fatigue variety for the remainder of the article," which reflects the current deep-rooted confusion in the terminology.

We consider it unnatural to categorize both fatigue fractures and insufficiency fractures as the single entity of "stress fracture," because the two are different from a biomechanical viewpoint. In terms of the stress-strain curve based on Wolff law, a fracture occurs when stress (ie, external force) exceeds the failure point (ie, intrinsic strength) of bone. An acute traumatic fracture occurs when an abnormally large external force is applied to a bone with normal strength, while a pathologic fracture occurs when a minimal external force is applied to a bone of diminished intrinsic strength (owing to weakening factors such as tumor or infection). Both fatigue and insufficiency fractures are also caused by such "minimal" forces. In the latter, the fracture occurs because the intrinsic strength of the host bone has already been diminished; in the former, the fracture occurs because repeated application of the minimal forces acts to reduce the intrinsic strength of the host bone. The two types of fractures thus differ in the nature of the decline in intrinsic strength: In an insufficiency fracture, the decline is irreversible and already present before application of the external force; in a fatigue fracture, the decline is reversible, but a crystalline failure of the affected bone is eventually induced through repeated application of the external force. As these differences suggest, these two fractures involve different biomechanical developments. In actuality, the insufficiency fracture has the same biomechanics as the pathologic fracture. The rationale for viewing insufficiency fractures and pathologic fractures separately may have been derived from the fact that a pathologic fracture due to a tumorous condition is similar to an acute traumatic fracture in terms of clinical course and management. However, not only pathologic fractures but also so-called insufficiency fractures due to osteomalacia or rheumatoid arthritis require management different from that of fatigue fracture, which occurs in basically normal bone. Thus, even from a clinical perspective, it remains difficult to justify the current usage of fracture-related terms, where "stress fracture" refers to both fatigue and insufficiency fractures while "pathologic fracture" refers only to tumor-induced fractures.

To better clarify this situation, we suggest that fractures be

classified into three categories: (a) traumatic fractures that occur in normal bone, (b) fatigue fractures that occur in transiently and reversibly weakened bone, and (c) insufficiency (or pathologic) fractures that occur in irreversibly weakened bone. We prefer to avoid the term "stress fracture" itself.

The definition and classification of fractures clearly involves a number of complicating factors. Given this complexity, an approach based on fracture mechanisms rather than phenomena may represent a solution offering considerable long-term clinical advantage.

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■ Metformin and Contrast Media: Genuine Risk or Witch Hunt?

From:

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Editor:

In Letters to the Editor in the November 1995 issue of *Radiology*, radiologists at two different centers expressed concern about contrast medium administration in patients with diabetes managed with metformin (Glucophage; Bristol-Myers Squibb, Princeton, NJ), a biguanide oral antihyperglycemic (1,2). Both noted that excretion of the drug is primarily renal. Metformin therefore accumulates in the setting of renal dysfunction. This in turn increases the known risk of biguanide-induced acidosis, which is fatal in 50% of cases. The manufacturer's package insert recommends discontinuation of the drug for 48 hours before and for 48 hours after contrast medium administration. One of our own radiologists saw the letters and relayed the warning to the faculty, technologists, and nursing staff. Eventually, risk management and hospital administration also took notice. Soon, a hospitalwide policy was adopted that mandated compliance with the package insert.

As a consequence, patients with poorly controlled diabetes are presenting for contrast-enhanced studies. Severe hyperglycemia and early ketoacidosis have been noted. We reviewed the literature to assess the actual risk. Our conclusion is that lactic acidosis after contrast-medium administration in patients with diabetes managed with metformin is extremely rare if pre-existing renal disease is excluded. The literature contains over 100 case reports of lactic acidosis, most (94%) in patients with pre-existing renal disease. The vast majority of these patients had not received a contrast medium. A contrast medium was implicated in only seven patients. Renal function before contrast medium administration could not be ascertained in all of these patients. However, let us assume that at least a few of the seven had normal renal function before contrast medium administration and developed contrast-induced renal dysfunction. With metformin, this scenario could lead to lactic acidosis. However, the incidence of contrast-associated renal failure, even in a diabetic population, is very low. More importantly, lactic acidosis occurs in only three per 100,000 patients per year due to metformin alone, without the implication of contrast media. If we take a little liberty with the statistics and multiply the overall incidence by the per-

centage of reported cases of lactic acidosis in diabetic patients receiving metformin who also receive contrast media (7%), we end up with a projected incidence of about two cases per million patients per year (ie, metformin plus contrast medium that leads to lactic acidosis). The incidence of fatal contrast medium reactions is higher. The literature on the biguanides, of which metformin is only one, suggests that age, alcohol use, and high dosage are the most important risk factors for lactic acidosis. Renal disease, cardiac disease, or both exacerbate these risks.

Why the overreaction? Before metformin, there was an unfortunate clinical experience with phenformin, another biguanide chemically related to metformin. Phenformin was also used to manage diabetes, but the associated high incidence of lactic acidosis resulted in its withdrawal from the market as a drug for diabetes in the 1970s. Metformin (like all biguanides) inhibits lactate metabolism, but to much less of an extent than phenformin. The phenformin experience has not been repeated with metformin, but the reputation of the earlier biguanide still influences manufacturers and doctors alike.

We withhold metformin before and after contrast medium administration in diabetic patients with renal dysfunction. However, we question the practice of routinely subjecting diabetic patients with normal renal function to several days of poor diabetic control or to a change in management (substitution of insulin). The legacy of phenformin, anecdotes, scattered case reports, product inserts, and medicolegal anxieties are more at the heart of this issue than scientific fact. The risks of metformin are overstated.

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■ Publishers' Row on the Internet?

From:

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Editor:

Keeping the departmental radiology library up to date is a tedious but important task, especially in teaching institutions. We use the printed Publishers' Row in *Radiology* on a regular basis to keep our library current and well balanced within the given budgetary constraints.

Although the (quarterly) Publishers' Row is an excellent service readers can use to keep track of newly published books across the entire specialty, it is to some extent cumbersome to use. Staff members in all subspecialties need to go through the entire listing to filter out recent books in their respective areas.

Our suggestion is to introduce an electronic parallel of Publishers' Row, to be published at the Radiological Society of North America site on the Internet. The "electronic Publishers' Row" could be made available as a listing with basic sort and select functions, such as sorting or selecting according to year of publication and subspecialty. Alternatively, it could be presented as a database, which can be transferred to the receiver's personal computer to be processed with database software. Attaching the book reviews of those books that have been reviewed would be of additional advantage.

RSNA responds:

Dr van Schaik's suggestion is under consideration.

Errata

"Mammographic Phantom Studies with Synchrotron Radiation" *Radiology* 1996; 200:659-663. Page 662, in Tables 3 and 4, the mean glandular dose values should be in millirads.

Table 3
Calculated Mean Glandular Dose from the Monoenergetic Source

Phantom Thickness (mm)	Energy (keV)				
	17	18	19	20	22
15	20	18	17	16	15
45	181	123	85	72	51
75	2,271	1,059	508	367	125

Note.—Glandular doses are in millirads. To convert to Systeme International d'Unites, 1 mrad = 10 µGy. Monoenergetic x rays were normalized to an exposure dose of 2.1 µC/kg (8 mR) to the film.

Table 4
Mean Glandular Dose from the Polyenergetic Source

Peak Kilovoltage	Phantom Thickness (mm)	Mean Glandular Dose (mrad*)
24	15	22
25	45	107
30	75	373

Note.—Polyenergetic x rays were normalized to an exposure dose of 2.1 µC/kg (8 mR) to the film.

*To convert to Systeme International d'Unites, 1 mrad = 10 µGy.

"Adrenal Masses: Correlation between CT Attenuation Value and Chemical Shift Ratio at MR Imaging with In-Phase and Opposed-Phase Sequences." *Radiology* 1996; 200:749-752.

Page 750, first column, the last sentence read as follows: "The decrease in signal intensity was measured on the in-phase and opposed-phase images by obtaining the lesion-to-spleen signal intensity ratio on the in-phase images and dividing by the lesion-to-spleen signal intensity ratio on the opposed-phase images." Dr Peter V. Quagliano of the Department of Veterans Affairs, Hunter Holmes McGuire Medical Center, Richmond, Va, has notified us of an error. The sentence should have read as follows: "A measure of the signal decrease was obtained from the in-phase and opposed-phase images by measuring the lesion-to-spleen signal intensity ratio on the opposed-phase images and dividing by the lesion-to-spleen signal intensity ratio on the in-phase images."

We thank Dr Quagliano for bringing this to our attention.