unable to protect the stomach; on the other hand, misoprostol, while affording good protection of both gastric and duodenal mucosa, causes frequent side effects on the intestinal tract. The conclusions of Taha and Sturrock, however, must be accepted with caution.

First of all in a recent paper on the subject they admitted that there was no significant difference in the prevalence of endoscopic abnormalities among patients taking NSAIDs alone or NSAIDs plus gold if the size of such lesions was not taken into consideration. The relation between the claimed gastroprotective properties of gold and Helicobacter pylori infection is also unproven. The role of H pylori in NSAID gastropathy is still debated, but most studies deny that the microorganism can promote or worsen NSAID injury.

The evidence quoted by Taha and Sturrock in favour of an inhibitory effect of gold on H pylori is mostly based on reports which have appeared only as abstracts. Full length papers provide conflicting data. Taha and Sturrock mention a possible methodological inconsistency, but negative results arose also in studies where, in addition to H pylori seroprevalance, the urea breath test was used. It must also be remembered that the possible bactericidal effect against H pylori by gold in vitro failed to be confirmed in vivo.

Finally, to infer that gold might exert gastroprotective effects similar to those of bismuth, merely because they are classified close to one another in the periodic table of elements, is mere speculation, especially if we consider some experimental reports of gold induced gastric mucosal lesions.

As a physician and a researcher concerned with the problem of preventing NSAID induced gastroduodenal lesions I do hope that eventually the hypothesis of Taha and Sturrock will prove correct. For the time being, however, this remains a (golden) dream that does not justify widespread use of gold in clinical practice as a possible prophylactic of NSAID gastropathy.

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7 Watanabe H. Pathogenesis of gastric mucosal damage induced by intraperitoneal administration of gold thioglucose in rats. Gastroenterology 1989; 84: 357-64.

**Author’s reply:** We read Dr Guslandi’s letter with interest and wish to make the following points:

1 Although he rightly points out that we have shown no overall significant difference in the prevalence of endoscopic lesions between patients receiving non-steroidal anti-inflammatory drugs (NSAIDs) alone, or together with gold, the prevalence of ulcers was different between the two groups. Superficial gastric erosions are commonly found in the group of patients that we examine by endoscopy, but there is no evidence that these erosions progress to ulcers, or cause serious ulcer complications, such as perforation.

2 There is still considerable controversy about the precise role of H pylori in mediating NSAID related damage. A recent leader from our group considered this controversy in some detail.

3 We certainly have not advocated the widespread use of gold as a means of prophylaxis of NSAID gastropathy, and our editorial highlights an interesting effect of gold on NSAID gastropathy, the mechanisms of which have still to be elucidated.

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**Correction**

**Acknowledgments to assessors in 1992** (Ann Rheum Dis 1993; 52: 248)

We regret that owing to the idiosyncrasies of the fax machine the following names were omitted from the list of assessors:

Holt M  
Morris C J
Hopkinson N  
Moskowitz R W
Hosking D  
Murphy G
Humphrey M  
Notarianni L

The editor would like to thank all the assessors for giving their time and help in 1992.
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