assume that all patients classified as experiencing emesis in the study actually aspirated. Therefore, the use of the term “aspiration” in the tables was imprecise. We apologize for the confusion.

It is not unusual during either standard CPR or IAC-CPR for patients to “regurgitate” stomach contents. Teleologically, one would expect a higher incidence of regurgitation in patients receiving abdominal compressions. However, the currently published experience with IAC-CPR does not bear this out. In our original study of IAC-CPR in 103 hospitalized patients, we found no difference in the rates of emesis between those patients receiving standard CPR and those patients receiving IAC-CPR. In an earlier out-of-hospital trial of IAC-CPR by Mateer et al.,3 there was no significant difference in the occurrence of emesis either before or after endotracheal intubation in 246 patients randomized to receive either standard CPR or IAC-CPR. In fact, data exist that suggest that IAC-CPR may actually lower the incidence of emesis during CPR. Babbs and coworkers4 demonstrated a significant reduction in gastric insufflation with the use of IAC-CPR in a canine model of cardiac arrest. They propose that because abdominal pressure is maintained during ventilation, IAC-CPR may actually decrease the chance of regurgitation by preventing gastric insufflation.

Data from published randomized studies report on several hundred patients who have received interposed abdominal compression during CPR. In addition, hundreds of animals have been subjected to the technique in the laboratory. To date, there has been only one published case report of a complication from the use of IAC-CPR.6 Although IAC-CPR appears to be safe and may lower the rate of gastric content regurgitation, the authors would not recommend its use in a patient with an unprotected airway. However, studies with IAC-CPR in patients who are not intubated are warranted at this time.

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References

High Stored Iron and the Risk of Ischemic Heart Disease

The contribution by Salonen et al.,1 with editorial comment by Sullivan,2 affirms that high iron levels promote ischemic heart disease (IHD). In their Finnish Study, in which a cohort of symptom-free men was followed for 3 years, those with serum ferritin ≥200 µg/L had a 2.2 risk factor-adjusted risk of acute myocardial infarction compared with men with a lower serum ferritin. Additionally, Sullivan2 noted that persons with high iron stores are more susceptible inter alia to infections, cancer, and aging. Accordingly, he called for a comprehensive reexamination of the bearing of iron intake, supplements, and stores on health and disease.

In a reappraisal, in search for a new paradigm, it must be kept in mind that conclusions reached from observations made on Western populations do not necessarily apply to contextually different Third World populations. In the present issue, we wish to mention the South African black population, who, until recently, had a high prevalence of siderosis or abnormally high iron stores, a phenomenon stemming from habitually high iron intake, mainly adventitious, derived from food prepared in iron vessels.3 In the extreme, concentrations (dry weight) of iron reached 2% in the liver and 5% in the spleen.4 A recent study revealed that about two thirds of rural elderly men and a third of elderly women had ferritin levels ≥200 µg/L.5 Yet, IHD remains uncommon.6 In rural areas, it is virtually absent (many blacks reach very old age). Even in large cities, the incidence in blacks, judging in part from hospital admissions, is less than a tenth of that among whites. This low rate is somewhat surprising, for their prevalence of hypertension is higher than that in whites; smoking is common and rising particularly among men; furthermore, the more affluent tend to have high serum cholesterol levels although additionally, they have high levels of high-density lipoprotein cholesterol. There are other interethic, puzzling situations in this particular field. Whereas in white schoolchildren, serum cholesterol rises with body mass index, it does not do so with black schoolchildren. Moreover, in black women, obesity (very common) has far less associations with hypertension, hyperlipidemia, and diabetes than is the case with obese white women.7

Among the Indian population in South Africa, iron deficiency with low iron stores, not wholly explicable, is common, especially among women.8 Yet, mortality rate from IHD is very high among Indian men and women, indeed, higher than the rate in the local white population9 (this also prevails concerning Asians in the United Kingdom).

We do not doubt that in the Western setting, high iron stores could be promotive of IHD. However, in contextually different populations, it would seem that high and low iron stores can have different connotations of risk regarding susceptibility to the disease.

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References

Reply
Walker and coworkers mention in their letter the South African black population, in which elevated body iron stores were common but incidence of coronary heart disease (CHD) was low. This observation is not in disagreement with our findings in eastern Finnish men, in which elevated serum ferritin concentration was associated with an excess risk of acute myocardial infarction only if the serum low-density lipoprotein cholesterol level also was high.1 Walker and coworkers mention that “the more affluent tend to have high serum cholesterol levels” but give no estimates of the general cholesterol levels. Most traditional rural African populations have been reported to have low serum cholesterol levels.2

We elaborated in our reply to Giles and coworkers3 our view that cross-population comparisons do not provide strong evidence for or against etiological hypotheses. One would not expect all populations to fit into an ecological correlation between a risk factor and a disease. This is an analogy with individuals in whom a perfect prediction of the risk is possible only if values of all etiologically relevant factors were known. One deviant risk factor value may either protect or expose an individual or a population and may make other risk factors appear less predictive, as in the case of the “French paradox.”4 However, it would be important to investigate the association between serum ferritin and CHD risk within a population with high overall iron stores and possibly a large range in iron stores such as the South African blacks.

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Thrombolysis as the First Line of Therapy for Cardiac Valve Thrombosis
In the January issue of Circulation, Silber et al2 reported the results of a prospective study of thrombolysis as first-line treatment for St Jude cardiac valve thrombosis. We agree with the authors of this study in that thrombolysis can be safely used as the initial treatment for thrombosed cardiac valves with excellent chances of success. However, we wish to point out that their statement “another 69 patients receiving therapy for cardiac valve thrombosis have been reported with satisfactory results” is not correct. In 1988, our group reported the successful treatment of a Björk-Shiley mitral valve thrombosis with high short-term doses of streptokinase (1 000 000 to 1 500 000 IU as a 1 to 3-hour intravenous infusion).2 In 1991 and 1992, we described five other patients treated in the same manner, three of whom had a satisfactory outcome.3,4 Two of these patients had Björk-Shiley mitral prostheses and the third a St Jude pulmonary valve. None of them suffered hemorrhagic complications. One patient with a Björk-Sorin mitral valve died from cardiogenic shock. The fifth had a Björk-Shiley tricuspid prostheses and had to have a valve re-exploration despite thrombolytic therapy. The pathological study revealed the formation of an extensive fibrous pannus over the “thrombosed” valve. This phenomenon has been reported to be a frequent finding when thrombolytic treatment is unsuccessful.5

In their bibliographic review, Silber et al did not mention these three reports that we feel support the conclusions that are drawn from their study.

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References

Reply
We thank Dr Martino and colleagues for pointing out these six additional cases in which thrombolytic therapy was used to treat prosthetic valve thrombosis. The 1- to 3-hour infusion of streptokinase used by Dr Martino and colleagues has the advantage of costing considerably less than the prolonged urokinase infusion used in our study. A study comparing the costs, success rates, and frequency of thromboembolic and bleeding complications of the alternative regimens (urokinase, streptokinase, tPA, and perhaps even simple heparin therapy) for thrombolytic therapy of prosthetic valves would be useful. However, such a trial would be difficult because of the infrequent occurrence of these events.

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High stored iron and the risk of ischemic heart disease.
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