Non-English Acronyms Have to be Explained in Their Native Languages

To read an article with unexplained acronyms is time-consuming and aggravating. To read an article with non-English acronyms explained in the English language is equally frustrating and befuddling.

Three articles2–4 in a recent issue of The American Journal of Cardiology (November 1, 1999) are such examples. The article on drug conversion of atrial fibrillation2 did not bother to explain the acronym PARSIFAL at all. The article on unstable angina reported3 that was given. However, had the authors explained the study in Spanish—Recursos Empleados en Tiempos de Espera5—there would not be any problem at all.

The other article on atrial fibrillation4 was reported by the RESCATE Study Group from Barcelona, Spain, explained RESCATE as Resources Used in Acute Coronary Syndromes and Delays in Treatment. I dare any of your readers to decipher the acronym from the English definition that was given. However, had the authors explained the study in Spanish—Recursos Empleados en el Sindrome Coronario Agudo y Tiempos de Espera5—there would not be any problem at all.

The other article on atrial fibrillation4 was reported by the SOCESP Investigators. Unfortunately, the authors explained SOCESP in English—The Cardiology Society of Sao Paulo. Since I do not know Portuguese, I am at a loss as to how SOCESP came to stand for the Cardiology Society of Sao Paulo.

I wish to make a plea again that explanations of any non-English acronym should be in the native language.5–7 Otherwise it would be utterly meaningless, almost as bad as not explaining it at all.

Tuong O. Cheng, MD
Washington, DC
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4. de Paola AAV, Veloso HH, for the SOCESP Investigators. Efficacy and safety of sotalol versus quinidine for the maintenance of sinus rhythm after conversion of atrial fibrillation. Am J Cardiol 1999;84:1033–1037.

Mechanism of Cardioprotective Effect and the Choice of Alcoholic Beverage

In most Western countries alcoholic beverages are an integral part of diets. They consist of about 4% to 6% of the average energy intake.2 Epidemiologic, experimental, and clinical investigations have proved that diets, supplemented with various kinds of alcoholic beverages, have a positive influence on coronary artery disease (CAD), improving lipid metabolism, increasing anticoagulant and antioxidant activity of consumers, and decreasing mortality from CAD.3–7 Therefore, we read “Type of Alcoholic Beverages and Risk of Myocardial Infarction,” by Gazzano et al8 with great interest. As was stated in their study, “...light-to-moderate intake (of alcoholic beverages) may lower all-cause mortality largely by a reduction in risk of coronary heart disease.” It is a well known fact. Less is known about precise mechanisms, by which alcoholic beverages reduce the risk of coronary heart disease and choice of alcoholic beverage. The authors of this study tried to give answers to these questions. We do not think that the answers are satisfactory. They have written: “This case-control study...suggests that the observed benefit of each beverage type is largely mediated by HDL...” This statement does not contribute to our understanding “...of the mechanisms by which alcoholic beverages reduce the risk of coronary heart disease.”

Recent investigations have demonstrated that the positive influence of alcoholic beverages is mainly connected to polyphenols of their dry matter.9,10 So, Gorinstein et al found that dry matter of different alcoholic beverages positively influences lipid metabolism and plasma antioxidant activity of rats, and alcohol-containing and alcohol-free beverages have an equal influence on plasma lipid levels and plasma lipid peroxides in rats.10,11 Serafino et al12 reported that alcohol-free red wine enhances plasma antioxidant capacity in humans. Carbonneau et al13 observed an improvement in the antioxidant status of plasma and low-density lipoprotein in subjects receiving a red wine phenolics mixture. The above-mentioned experiments on laboratory animals and investigations of humans are underlying the role of phenolics in alcoholic beverages. No doubt, the mechanisms by which alcoholic beverages reduce the risk of CAD include first, the influence of their antioxidant phenolic substances.

The type of used alcoholic beverages plays a very important role. To the rhetorical question of Klatsky and Armstrong,14 “do red wine drinkers fare best?” there is a clear-cut answer. As was shown by Abu-Amsha et al,15 the phenolic content of alcoholic beverages determines the extent of inhibition of human serum and low-density lipoprotein oxidation. As was shown by us, among 3 alcoholic beverages (red wine, white wine, and beer), the highest content of phenolics is in red wine and the lowest is in white wine, and this difference is significant.16 Frankel et al showed inhibition of oxidation of human low-density lipoprotein...
by phenolic substances in red wine. Mosinger18 demonstrated that polyphenolics of red wine protect serum low-density lipoprotein against atherogenic modification. Serafini et al.12 reported that alcohol-free red wine enhances plasma antioxidant capacity in humans. According to Renaud and Lorgeril,1 consumption of red wine explains the French paradox for CAD. However, the authors of this study indicated that “...data on the specific type of wine were not adequate to distinguish red from white.” Therefore, their conclusion that “subjects preferring wine were found to have a significantly lower risk of death from coronary artery disease” cannot be considered correct. Red wine affects lipid metabolism, and antioxidant and anti-oxidant activities in consumers significantly more than white wine.10–13 Therefore, the authors of this very interesting article would be correct in also using antioxidant capacity to mediate benefits of different types of alcoholic beverages. It is clear that without investigation of the content of these antioxidants it is impossible to speak about types of alcoholic beverages and risk of myocardial infarction.

Shela Gorinstein, PhD
Abraham Caspi, MD
Immanuel Libman, MD, PhD
Simon Trakhtenberg, MD, DSc

Jerusalem, Israel
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Aortic Valve Sclerosis and Aerobic Exercise

Aortic valve sclerosis is detected by echocardiography as thickening and calcification of the valve leaflets. It is associated with increased mortality in the elderly and may indicate coronary disease.1 The primary cause, however, is probably cumulative mechanical stress, as indicated by the patterns of calcification2 and the close correlation with advancing age.3 The known hemodynamic effects of aerobic exercise magnify mechanical stress on the leaflets.

Aerobic exercise raises systolic, but not diastolic, aortic pressure4 and shortens the cardiac cycle, with quicker ventricular relaxation.5 Thus, aerobic exercise increases the magnitude and slope of the late systolic, retrograde, transvalvular pressure gradient. This distends and closes the leaflets faster and more forcefully, as reflected in a louder second sound.6 Beyond a critical threshold, increased force and more rapid flexing must strain the leaflets. Injury would be greater if aging has weakened the tissues.

Available literature is insufficient to establish a relation between aerobic exercise and valve sclerosis. Echocardiography of a 33-year-old runner with acute gout, however, discovered an aortic valve leaflet nodule, apparently a tophus, and the only one found.7 Tophi occur at sites of heavier mechanical stress, and a valve leaflet tophus is an “extreme rarity,” as stated in the report. This unusual finding, particularly at an early age, therefore suggests that the leaflet was injured by the patient’s hard training and 10-mile racing, a race distance more intense than the marathon.

Exercise histories are needed to test this hypothesis. If aerobic exercise is a factor, its intensity would be most important. Exercisers, particularly if older, could limit the intensity of their exercise to keep its pressure and hear rate effects at safer levels.

Herbert W. Copeland, MD
Boca Raton, Florida