New evidence favoring the Mediterranean diet

Many population-based studies and clinical trials have demonstrated the benefits of the Mediterranean diet (MD), rich in fruit, vegetables, legumes, whole grains, olive oil, moderate alcohol and little meat on adverse cardiovascular events. At the same time, consumption of saturated fats, refined carbohydrates, and alcohol in excess have been associated with poor outcome. But, which is the weight of each dietary pattern in the outcome of patients, when “good” and “bad” food is consumed in daily life? The STABILITY trial evaluated the effect of Darapladib, a specific inhibitor of lipoprotein-associated phospholipase A2 in patients with stable coronary artery disease [prior myocardial infarction (MI), prior coronary revascularization and multivessel disease] with at least one risk factor (age >60 years, diabetes, low HDL-cholesterol levels, glomerular filtration rate between 30 and 60 mL/min per 1.73 m2 or urine albumin:creatinine ratio >30 mg/g). Although the study did not demonstrate any benefit related to the drug, the following information is interesting. At the beginning of the study, the participants were asked about their dietary habits. Participants were asked how many times during a week they consumed servings of red meat, fish, whole grain and refined grain products, diary products, legumes, vegetables, fried food, eggs, sugar sweetened beverages and alcohol. Two scores (S) were constructed for each patient: the Mediterranean diet score (MDS) and the Western diet score (WDS). In each case, the highest S reflected higher consumption of the corresponding food.

Data were obtained from 15,482 patients. Median MDS was 12. Subjects with higher MDS were less likely to be current smokers, took slightly more physical activity, had lower white blood cell count, C-reactive protein and fasting blood glucose levels. On the contrary, the WDS was only associated with fasting blood glucose levels and diabetes. After 3.7 years of mean follow-up, MDS scores >12 were associated with lower incidence of major adverse cardiovascular events (MACE: cardiovascular death, non-fatal MI or non-fatal stroke). MACE occurred in 10.8% of study participants with MDS ≤12, 10.5% of subjects with an MDS of 13–14 and 7.3% of subjects with MDS ≥15; one unit increase in MDS above 12 was associated with a 7% risk reduction (95% CI, 4-10). In a multivari-
is achieved matters or not. We have read many publications about the statin hypothesis, referring to the preferential effect achieved with these agents. On the contrary, other voices state that the effect achieved on MACE does not depend on the agent but on the reduction of LDL-C levels. A recent meta-analysis represents the greatest effort published so far to answer this question. The meta-analysis considered 49 randomized trials (with a total of 312,175 patients during a mean follow-up period of 4.3 years) of lipid-lowering agents or interventions, with at least 6-month duration and 50 MACE or greater, including myocardial infarction (MI).

Studies of nine different types of LDL-C reduction approaches were included and were divided into 4 groups: a) statins (25 studies); b) nonstatin therapies that produce LDL receptor expression upregulation (8 studies of diet, bile acid sequestrants, ileal bypass surgery, and ezetimibe); c) interventions that reduce LDL-C levels by other mechanisms (15 studies of fibrates, niacin and CETP inhibitors); and d) PCSK9 inhibitors, which upregulate LDL-C clearance through the LDL receptor but for which dedicated cardiovascular outcome trials have not yet been completed.

For the statin trials, each 1-mmol/L (38.7 mg/dL) reduction in LDL-C levels was associated with a RR of 0.77 (95% CI, 0.71-0.84) for MACE, without significant difference between primary (RR 0.70) and secondary (RR 0.79) prevention. In the case of group b) interventions, each 1-mmol/L reduction in LDL-C was associated with an RR of 0.75 that was not statistically significant compared with statins. Combining the data from all the 33 aforementioned trials generated the meta-regression line (predicted RR of MACE for various levels of LDL-C reduction between the control group and the intervention group). The observed RR of MACE for each of the five different interventions was within 2% of the predicted value from the regression line normalized to the magnitude of LDL-C reduction.

In the case of niacin, the observed RR of 0.94 for MACE was similar to the expected RR of 0.91 according to the magnitude of LDL-C reduction achieved. The observed RR of 0.88 for fibrates was lower than the expected RR of 0.94, meaning a greater risk reduction beyond LDL-C reduction. On the contrary, for CETP inhibitors the observed RR was 1.01 and the expected RR was 0.90, (absence of risk reduction). Finally, and based on the information available up to the present time, PCSK9 inhibitors had an estimated RR of 0.49 versus an expected RR of 0.61.

This meta-analysis (of studies, not of individual data, which would have been optimal) supports the LDL hypothesis: statins, but also therapies that ultimately work predominantly through upregulation of LDL receptor expression, are associated with MACE reduction. We have already mentioned that in the IMPROVE-IT trial, the RR reduction for MACE with ezetimibe was similar to the RR expected for the level of LDL-C achieved. For the case of the other interventions, some considerations can be made. The reduction of events observed with niacin, though low, corresponds with the reduction of LDL-C. The reduction achieved with fibrates, which was somewhat greater than the one expected only by the effect on LDL-C can be explained by the additional effect on triglycerides. On the contrary, the lack of reduction of events observed with CETP inhibitors, despite lowering LDL-C levels, seems to be associated with adverse events, particularly with the activation of the renin-angiotensin system. We should wait until the results of large randomized trials with PSCK9 inhibitors specially designed to evaluate the effects on MACE are published. In any case, and always, it should be borne in mind that for a given relative risk reduction, the absolute reduction of events will always depend on the baseline risk.

Hypothyroidism is rarely considered for the evaluation of cardiovascular risk


Thyroid abnormalities have been associated with higher risk of cardiovascular events. Overt hypothyroidism is more common in older women and is associated with higher incidence of coronary artery disease and heart failure. The risk associated with subclinical hypothyroidism is a matter of discussion, as although it has been related to adverse outcome in observational studies, it might have a protective role over the age of 85. We present a recent study conducted by the Mayo Clinic that explored the association between hypothyroidism and the incidence of events in a population of patients with coronary artery disease undergoing percutaneous coronary intervention.

Between 1994 and 2008 25,317 patients underwent percutaneous coronary intervention. A group of 2,430 patients with no prior myocardial revascularization surgery or history of malignancy, a new coronary angiography during follow-up and TSH data >0.3 mU/ml to exclude hyperthyroidism was selected. Patients were divided into three categories: euthyroidism (E, with TSH values between 0.3 and 0.5 mU/mL, n=1,835), subclinical hypothyroidism, (SCH, TSH values >5 and <10 mU/ml, n=319) and overt hypothyroidism (OH, TSH ≥10 mU/ml, n=276). Patients with SCH or OH were further stratified according to those receiving no thyroid replacement therapy (TTR/T) in patients who had adequate TRT (TSH between 0.3 and 5 mU/mL), and patients with inadequate replacement on TRT (TSH <0.3 or >5 mU/ml).

Compared with the E group, patients with SCH or OH were significantly older and more likely to be female (54% vs. 29%), or to have diabetes, hypertension and heart failure. The prevalence of left main coronary
artery disease and three-vessel disease was similar among the three groups, but the prevalence of one-vessel disease was higher in patients with SCH or OH. There were minimal differences in medical treatment, and patients with hypothyroidism were more likely to be taking nitrates, ACE inhibitors and amiodarone (3% vs. 1%). During hospitalization, the incidence of heart failure was significantly higher in patients with hypothyroidism; however, the frequency of cardiovascular events did not differ significantly between groups. After adjusting for age, sex, risk factors, number of vessels, type of stent and treatment, the risk of MACE (HR: 1.28, 95% CI, 1.13-1.45), myocardial infarction (HR:1.25), heart failure (HR: 1.46), need for new re-vascularization (HR: 1.26), and stroke (HR: 1.62) was higher in the hypothyroid group compared with the euthyroid group at the 10-year follow-up. There were no significant differences between SCH and OH. For patients on adequate TRT, the rate of events was lower compared with patients taking no TRT and patients with inadequate TRT. The coronary angiographies of a random sample of 102 patients with hypothyroidism and 306 with E were compared during follow-up, demonstrating a higher progression of coronary artery disease in patients with hypothyroidism.

This observational study confirms the adverse outcome of hypothyroidism in a specific cohort. Part of this phenomenon can be explained by the different pattern of covariates that the multivariate analysis tries to correct. However, there is still the probability of residual confounding: the presence of variables not accounted for in the analysis and related to hypothyroidism that may be determinants per se of the poor outcome. Another point that makes the conclusions of this study weaker is the presence of a selection bias: the patients included were less than 10% of the population undergoing PCI during that period, those who had TSH levels assessed at the time of PCI. One can ask oneself about the reasons of measuring TSH levels in those patients in particular, and whether this did not already reflect a population with specific characteristics. We should also add that T4 and T3 levels were not measured, which would have better characterized the endocrinological condition. Finally, thyroid replacement therapy was not indicated at random, and that the lack of replacement therapy can also mean that these patients were not well-treated in other aspects and, thus, had worse outcome. In any case, this paper calls the attention about a condition that is not always considered, and is an invitation to carry out prospective clinical trials.

The DANISH study: Is there a decrease in the indication of defibrillator devices in patients with non-ischemic etiology?

Although the implantation of implantable cardioverter defibrillator (ICD) devices in patients with nonischemic heart failure has not demonstrated a significant reduction of mortality in individual studies, the meta-analysis by Desai et al. (2004) justified the similarity of these patients with the indication in patients with ischemic heart disease. The results of the DANISH trial, which have been recently published, question that finding.

The trial enrolled patients with nonischemic dilated cardiomyopathy (without significant coronary artery disease or with one or two-vessel coronary artery disease if the extent of coronary artery disease was not considered to be sufficient to account for left ventricular dysfunction), left ventricular ejection fraction (LVEF) ≤35%, NYHA class II or III, or NYHA class IV if cardiac resynchronization therapy was planned and under optimal medical treatment. Participants were randomly assigned in a 1:1 ratio to either the ICD group or the control group. The enrollment started in 2008 and included 1,116 patients; median age was 63 years, 53% were in FC II, 45% in FC III and the rest were in FC IV. Median LVEF was 25%. Median QRS duration was 146 ms and cardiac resynchronization therapy was indicated in 58% of the cases. In a median follow-up period of 67.6 months; the incidence of all-cause death (the primary endpoint) was 21.6% in the ICD group and 23.4% in the control group (HR 0.87, 95% CI, 0.67-1.12). There were no significant differences in the incidence of cardiovascular mortality, but cardiac sudden death was significantly lower (4.3% vs. 8.3%, HR 0.50, 95% CI, 0.31-0.82). The results of subgroup analyses showed a significant interaction with age: the use of ICD produced a significant reduction of 36% in all-cause death among patients younger than 68 years of age. There was a trend toward interaction (p=0.06) with NT-proBNP levels, with a significant reduction in levels <1,177 pg/ml.

The DANISH trial included a peculiar population, with high incidence of indication of cardiac resynchronization therapy and very good treatment (92% with beta-blockers, 97% with renin–angiotensin system inhibitors or angiotensin receptor blockers and almost 60% with mineralocorticoid-receptor antagonists). The results of this study suggest that the universal use of ICD in primary prevention for patients with nonischemic heart failure under optimal medical treatment does not improve the prognosis. But, we add, these results also indicate that the indication should at least be considered in younger patients with less involvement (in whom the risk of sudden death is proportionally higher). Even being the result of subgroup analysis, this conclusion is plausible. The SAC Consensus Statement of Heart Failure, recently presented at the 42nd Argentine Congress of Cardiology, recommends ICD implantation for primary prevention in patients with ischemic heart failure, LVEF 33% and FC II-III (class I, Level of evidence A), and in patients with nonischemic heart failure (class IIa, Level of evidence B).
Higher rate of events, a corollary of the poor adherence to treatment in cardiovascular disease

Despite over the past decades substantial therapeutic advances have been achieved in the field of cardiovascular diseases, attaining favorable outcomes depends on the correct implementation of such advances. Patient adherence to medical prescriptions is a crucial aspect (but not the only one). Lack of adherence to the treatment indicated must impact on the expected outcomes. But to what extent? An observational study recently published expresses such effect in numbers.

Two cohorts were defined from the databases of an important health insurance company from the United States. The first cohort included patients who initiated both statin and angiotensin-converting enzyme (ACE) inhibitor medications after hospital discharge for myocardial infarction (MI) between January 2010 and February 2013, with data 6 months before and after the MI. The discharge date of MI hospitalization was identified as the index date. The second cohort was made up of outpatients who initiated both statin and ACE inhibitor medications and also had atherosclerotic disease in at least two different territories or a revascularization procedure in any of them between January 2010 and December 2011. The first statin and ACE inhibitor prescription was identified as the index date. The dates of the subsequent prescriptions were available for each patient, as well as the information about their outcome. In both cases, the follow-up period was through December 2013. In each case, the proportion of days covered (PDC) by the medication was estimated, and was calculated as the number of days between the first fill and the end of the follow-up period divided by the number of days covered by the prescription fills during the follow-up period (6 months for the post-MI cohort and 12 months for the atherosclerosis cohort). Patients were categorized into 1 of 3 groups on the basis of their PDCs: fully adherent (FA, PDC ≥80%); partially adherent (PA, PDC 40% to 79%); and nonadherent (NA, PDC <40%). The primary endpoint was the occurrence of major cardiovascular events (MACE), which included all-cause mortality, nonfatal MI, nonfatal stroke, or any coronary revascularization.

The post-MI cohort included 4,015 patients: 43% FA, 31% PA and 26% NA. Mean PDC was 93% for FA, 62% for PA, and 21% for NA. The group with the worst profile of risk factors was the FA category. The incidence of MACE during follow-up was 18.9% for PA, 24.7% for FA and 26.3% for NA. Multivariate analysis, which considered clinical and demographic variables, comorbidities, other medication and use of medical services, revealed an adjusted HR for the FA group of 0.81 (95% CI, 0.69-0.96) compared with the PA group and 0.73 (95% CI, 0.61-0.87) compared with the NA group. There were no differences between the PA group and the NA group. The reduction in the number of hospitalizations was significant in the FA group compared with the other two groups.

The atherosclerosis cohort included 12,976 patients: 34% was FA, 31% was PA and 26% was NA. Mean PDC was 90% for FA, 62% for PA, and 19% for NA. The profile of risk factors was progressively worse as adherence was lower. The incidence of MACE during the 2-year follow-up was 8.4% for FA, 12.2% for PA and 17.2% for NA. Adjusted HR was 0.76 for the FA group compared with the PA group and 0.56 compared with the NA group. In this case, the PA group had better outcome than the NA group (HR 0.73). A sensitivity analysis was also performed excluding MACE that occurred during the first year (the adherence assessment period) and showed significant differences for the FA group compared with the NA group, but not with the PA group. In both cohorts, FA to the indications was associated with lower costs due to hospitalizations and procedures.

The results of this study confirm some presumptions and information from other registries: full adherence to indications (and we are only talking about compliance of two drugs with recognized effects on the events!) is usually low. In the case of the period of time after an acute coronary event, adherence occurs in about 40% of the cases. And, based upon this information, only full adherence ensures better outcome. Partial adherence is not different from non-adherence. Being fully adherent reduces the risk of MACE by more than 25% compared poor adherence. In the context of chronic atherosclerosis, the incidence of MACE is lower, but a gradient is evident: the greater the adherence, the better the outcome. The reasons for the lack of adherence to the medication prescribed are multiple and, as we already know, depend on the health care system, patients, and even on the attending physicians. Defining these barriers and breaking them down is undoubtedly more important than looking for alternative treatments in expensive trials, when those treatments that have already been proven and validated are not used as they should.

Atrial fibrillation, precedents and consequences over the time

Major risk factors for atrial fibrillation (AF) are well-known and include age, hypertension (HT), diabetes mellitus (DM), smoking, obesity, and history of cardiovascular disease and heart failure (HF). In addition,
AF is an established risk factor for HF and stroke. The recently published ARIC study, a biracial, prospective cohort study of cardiovascular disease and atherosclerosis risk factors performed in the United States, reported the association between AF risk factors, the development of AF and the outcome of patients over the time. The ARIC study included 15,792 men and women of 45 to 64 years of age between 1987 and 1989. After the initial assessment, study participants were examined in 4 additional visits between 1990 and 1992, 1993 and 1995, 1996 and 1998 and finally between 2011 and 2013).

To define the association of AF with the prevalence of risk factors preceding AF diagnosis and the subsequent development of cardiovascular outcomes, 2,456 individuals with AF diagnosed during the follow-up period were included and were matched with 6,414 control subjects without AF by age, sex, race and center. Index date was defined as the date of AF diagnosis for each case and the same date for the corresponding matched controls. As the time of AF diagnosis is usually imprecise, follow-up time was categorized in 5-year periods: \(-17.5\) years, \(-17.5\) to \(-12.5\) years, \(-12.5\) to \(-7.5\) years, \(-7.5\) to \(-2.5\) years, \(-2.5\) to 2.5 years (reference category), and >2.5 years. The index date was defined as \(t = 0\). The prevalence of risk factors and cardiovascular outcomes [HF, stroke, and myocardial infarction (MI)] was defined in each time period separately for AF cases and controls. In this way, “trajectories” were defined over time for each cardiovascular outcome. The authors reported that: a) the prevalence of risk factors was greater for patients with AF across all the time periods; b) the prevalence of HT and DM had a linear and parallel increase over time in AF cases and in controls up to and after the index date, with no inflexion point for AF; c) the prevalence of smoking and obesity increased slightly during follow-up but started to decline around the index date; d) on the contrary, the prevalence of MI, stroke and HF in AF cases had a J-shape pattern, with low prevalence before AF and steep increases in prevalence during the period of time close to AF diagnosis.

Another analysis considered 10,559 participants who participated in the first 4 visits and were free of AF at visit 4. As the trajectories of each risk factor and the outcome were known, the authors could establish patterns and define their ability to predict AF from the fourth visit. After a median follow-up of 15 years (between visit 4 and visit 5) participants with prevalent risk factors and those who had had them for a longer interval had increased risk of AF in a multivariate analysis adjusted by the rest of the risk factors, age, sex and race.

This analysis of the ARIC study confirms, in an elegant and applicable way, the association of AF with multiple risk factors and related clinical conditions. Hypertension and DM predispose to the development of AF but this does not modify the linear increase in the prevalence of both conditions afterwards. These chronic conditions are risk factors for AF but are not modified once AF develops. On the contrary, stroke, MI and HF also increase the risk of AF but the presence of AF also increases the risk of developing these conditions. The prevalence and the long duration of these factors and chronic conditions are associated with higher risk of AF and, thus, with adverse outcomes. The association of two of these conditions or greater should call the attention to look for AF. Knowing these “trajectories” over time (as the authors of the study call them) and the risk associated can make us adopt a more active attitude toward treatment and surveillance.

Obstructive sleep apnea: inconclusive evidence for CPAP


Obstructive sleep apnea (OSA) is associated with an increased risk of hypertension, diabetes, cardiovascular and cerebrovascular events. Obstructive sleep apnea causes activation of the sympathetic nervous system, inflammation and endothelial dysfunction, and produces important variations of intrathoracic pressure which may contribute to explain these phenomena. Observational studies and some randomized trials have demonstrated that the use of continuous positive airway pressure (CPAP) can reduce blood pressure, improve endothelial function and decrease insulin resistance. The SAVE study was designed to evaluate the effectiveness of CPAP in reducing the rate of major cardiovascular events among patients with moderate-to-severe OSA. The results of this study were recently presented at the European Society of Cardiology Congress.

The SAVE study was an international, randomized, parallel-group, open-label trial, with blinded end-point assessment. Eligibility criteria included age between 45 and 75 years with diagnosis of coronary artery disease or cerebrovascular disease and OSA that was established with the use of a home sleep-study screening device which determined the degree of sleepiness and blood oxygen saturation level. Patients were included in the study if the number of times per hour during the oximetry recording that the blood oxygen saturation level dropped by \(\geq 4\)% from baseline was of at least 12. The self-administered Epworth Sleepiness Scale which evaluates the degree of sleepiness the patient reports in certain situations was used to avoid including patients with symptomatic severe OSA; scores \(>15\) (the scores range from 0 to 24) were exclusion criteria. Patients with oxygen saturation \(<80\%\) for \(>10\%\) of the recording time or those with a pattern of Cheyne–Stokes respiration were also excluded. Potential participants were required to have a minimum level of adherence to CPAP therapy, which was defined as an average of 3 hours per night, dur-
ing a run-in period in which sub-therapeutic CPAP was used. The patients were randomly assigned to receive CPAP therapy plus usual care or usual care alone. Randomization was performed with the use of a minimization procedure to balance the group assignments according to site, type of disease and Epworth Sleepiness Scale score (<11 vs. ≥11). Although the original plan was to recruit 5,000 patients, challenges in achieving recruitment targets and statistical issues prompted to reduce the number to 2,500. With this sample size, the study would have 90% statistical power to detect 25% reduction of the primary composite cardiovascular end point (death, non-fatal myocardial infarction, nonfatal stroke, hospitalization for heart failure, acute coronary syndrome or transient ischemic attack) with CPAP.

A total of 5,844 patients met the initial eligibility criteria and moderate-to-severe OSA was confirmed in 3,246. Finally, 2,687 patients were included in the intention-to-treat analysis and were randomly assigned to receive CPAP plus usual care (1,346 patients) or usual care alone (1,341 patients). Mean participant age was 61 years, and 81% were men; more than 50% had coronary artery disease and the rest had cerebrovascular disease. Almost 80% had hypertension, 30% had diabetes and 15% were smokers. The mean body mass index (BMI) was 29, the mean oxygen desaturation index was of 28 events per hour, and the apnea–hypopnea index (the number of apnea and hypopnea events per hour of recording) was 29. The mean duration of CPAP therapy adherence in the first month of treatment was 4.4 ± 2.2 hours per night, which decreased to 3.5 ± 2.4 hours per night by 12 months and to 3.4 ± 2.3 hours thereafter. Mean follow-up was 3.7 years. The apnea–hypopnea index during CPAP use decreased to 3.7; 42% had good adherence to treatment (≥4 hours per night).

But, in fact, there were no significant differences in the primary end point: 17% in the CPAP group vs. 15.4% in the usual care group (HR 1.1, 95% CI, 0.91-1.32). There were no differences in age, sex, diabetes, baseline conditions, BMI or severity of OSA. Non-significant differences between groups were found for any of the cause-specific or composite end point, or considering only patients with good adherence to CPAP therapy and comparing them with patients of the usual care group with similar baseline characteristics. CPAP therapy was associated with a reduction in the Epworth Sleepiness Scale score (-2.5) and better scores of quality of life, anxiety and depression compared with usual care.

For different reasons related to pathophysiology, OSA, mostly in its severe form, has been associated with greater incidence of cardiovascular events. However, the randomized trials published up to the present have failed in demonstrating any benefit with non-invasive ventilation. The SAVE trial, with the highest number of patients ever included but follows the same pathway. An argument expressed in previous publications referred to adequate treatment adherence to achieve the expected effect. This study failed to demonstrate this effect even in patients adherent to CPAP therapy; they only presented improvement in quality of life, incidence of anxiety or depression. These effects should not be underestimated in a context in which only the reduction of hard endpoints seems to justify an intervention. But, surely, the results oblige to make a better selection of the population in which CPAP therapy is cost-effective. The question is why the intervention failed to achieve to effect expected. Are the mechanisms explaining OSA with poor outcome not so important? Is OSA a risk marker but not a risk factor? Could other intervention be more effective?

Predictors of mortality in acute myocardial infarction: A new and easy model


Although different risk models have been developed to predict mortality in patients hospitalized for ST-segment elevation or non-ST-segment elevation myocardial infarction (STEMI or NSTEMI), few have included a representative sample from routine clinical care. Among them, a risk model was developed and published in 2011 using data from the ACTION Registry–GWTG which included patients from more than 300 hospitals in the United States. Since then, the ACTION Registry–GWTG model has been expanded to identify patients with cardiopulmonary arrest as a form of presentation. In addition, the authors of this model constructed a risk model calculated for prospective risk stratification soon after patient presentation. We are presenting these advances.

Between January 2012 and December 2013, a total of 254,066 patients with NSTEMI and STEMI from 665 participating hospitals were included in the registry. For the present report, patients were excluded if they were transferred out of participating hospitals, leaving a final sample of 243,440 patients. This cohort was randomly divided into a derivation cohort (60%) to build the model and a model validation cohort (40%). Mean age was 64.6 years, 65% were men, 33% were diabetic, 74% had hypertension and 61% had dyslipidemia. Thirty-nine percent of the cases presented with STEMI, 4% presented after cardiac arrest, 4% in cardiogenic shock and 13% with heart failure. Demographic, clinical, laboratory and treatment variables were considered in relation to global mortality. In the multivariate analysis, the variables that were independently associated with in-hospital mortality were: age, STEMI, heart rate and systolic blood pressure, cardiac arrest, cardiogenic shock and heart failure at presentation, and creatinine clearance; and troponin ratio (baseline troponin value divided by the upper limit of normal).
The risk model assigns the following scores: a) for age, from 0 in < 40 years to 20 in >90 years; b) for systolic blood pressure (SBP), from 0 if SBP is >200 mm Hg to 19 if SBP is ≤90 mm Hg; c) for heart rate (HR), from 0 if HR is <40 bpm to 9 if HR is >150 bpm; d) for creatinine clearance (CrCl), from 0 if CrCl is >95 ml/min/1.73 m2 to 15 if CrCl is <30 ml/min/1.73 m2 or if the patient is in dialysis; e) for troponin, 0 if troponin ratio is <10 up to 3 if troponin ratio is >30; f) presentation after cardiac arrest: 14 points; g) presentation in cardiogenic shock: 13 points; h) presentation with HF: 5 points; i) presentation with STEMI: 5 points.

In-hospital mortality associated with scores <30, 30-39, 40-49, 50-59 and >59 was 0.4%, 1.7%, 5.5%, 18.5% and 49.5%, respectively. The area under the ROC curve for the model was high: 0.88.

Although we already have risk scores for acute MI, as the TIMI risk score and the GRACE score, the model here presented was constructed using a more recent cohort of patients and incorporates presentation after cardiac arrest as the most important news. This study has some limitations: the ACTION Registry–GWTG is a voluntary registry and we cannot trust the consecutive character of the patients; the participating hospitals are more likely to have catheterization laboratories; patients transferred out of the participating hospital were excluded; the registry only considers in-hospital mortality; the model has not been validated on an external dataset. Yet, the model has strong advantages: the number of patients analyzed and the variables used to construct it, which can be easily accessed. As we always say, prediction is always easier and more reliable for populations than for the individual patient. The use of this type of tools will never provide us certainty about the patient’s destiny, but can contribute to make us think about his/her possible outcome and, thus, indicate a more or less aggressive treatment.