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years (SD 2.0 years), 944 subjects had suffered a first CHD or stroke events, respectively 260, 218, 249 and 217 at 2, 4, 7 and 10 years of follow-up, and 1700 had died. After adjustment for socio-demographic variables, vascular risk factors, impairment in daily life activities and antidepressant use, the presence of DS was associated with a significant 31% increased risk of mortality (HR=1.31;95% CI: 1.15–1.48), while occurrence of a vascular event was related to a three-fold increased risk (HR=2.97; 95% CI: 2.56–3.44). There was no interaction between the presence of DS at study visits and occurrence of vascular event for the risk of mortality (p=0.50).
Conclusion: In older participants, the relative increased risk of all cause mortality associated with the presence of DS is independent of the occurrence of incident vascular events.
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965 | BEDSIDE
Temporal trends in the treatment and outcomes of septua-, octo-, and nonagenarians with acute coronary syndrome

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Background: Old patients with acute coronary syndrome (ACS) are a growing demographic with higher risk of worse outcomes than younger patients.
Purpose: To determine whether treatment and outcomes of old ACS patients changed over time.
Methods: We analyzed 13,662 ACS patients ≥70 years enrolled in the Acute Myocardial Infarction in Switzerland (AMIS) cohort between 2001 and 2012. Use of guideline-recommended therapies and in-hospital outcomes were analyzed according to three 4-year periods (2001–2004, 2005–2008, 2009–2012). To determine associations between use of percutaneous coronary interventions (PCI) and in-hospital mortality, logistic regression providing odds ratios (ORs) and 95% confidence intervals (CIs) was used.
Results: Between first and last 4-year period, PCI use increased from 43.8% to 69.6% of older ACS patients (P<0.001). The highest relative increase was found for primary PCI use among nonagenarians with ST-elevation myocardial infarction (3.6-fold increase between first and last 4-year period, P<0.001). Use of guideline-recommended drugs as well increased. At the same time, in-hospital mortality of the overall population decreased from 11.6% in the first to 10.0% in the last 4-year period (P=0.020), and in-hospital major adverse cardiac and cerebrovascular events from 14.4% to 11.3% (P<0.001). The highest relative decrease of in-hospital mortality (22.7%) between first and last 4-year period was observed among octogenarians (P=0.005). In the overall population, PCI use was associated with lower odds of in-hospital mortality and ORs did not markedly change between first and last 4-year period (adjusted OR for PCI use vs. no PCI use 0.29, 95% CI 0.22–0.40, in 2001–2004; and, adjusted OR for PCI use vs. no use 0.26, 95% CI 0.20–0.35, in 2009–2012).
Conclusions: Use of guideline-recommended therapies for ACS increased and in-hospital outcomes improved over the observed 12-year period. PCI use was associated with lower odds of in-hospital mortality with similar ORs between first and last 4-year period. This study suggests that better guideline adherence improves in-hospital outcomes of older ACS patients.

966 | BEDSIDE
Do risk factors explain the sex/gender gap in mortality from coronary heart disease?

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Background: In Europe, per year, approximately 253,000 men, but only 77,000 women die prematurely from coronary heart disease (CHD) before the age of 65, while, when considering all ages, slightly more women do so than men. CHD rates increase with age, however to a varying extent between men and women. At younger ages, incidence and mortality are markedly lower in women, whereas with increasing age this gap narrows. However, little is known regarding the contribution of cardiovascular risk factors to this sex/gender effect.
Purpose: While there have been studies investigating the possible different role of cardiovascular risk factors in men and women, there have not yet been, to our knowledge, any attempts to explore how much of the sex/gender effect is mediated through risk factors. Presumably, since no appropriate statistical modelling approach for survival data was available. Recently, a new approach for mediation analysis was developed that allows to assess the specific contribution of risk factors explaining the difference between men and women regarding CHD outcomes.
Methods: The sex-specific CHD mortality was examined in prospective cohort data from Austria, consisting of 117,264 individuals younger than 50 years (as a proxy for menopausal status) and 54,998 older ones, with 3,892 deaths from CHD during a median follow-up of 14.6 years. Mediation analysis was used to decompose the sex/gender effect into a direct and an indirect component that is mediated by the four major cardiovascular risk factors systolic blood pressure, total cholesterol, fasting blood glucose, and smoking status.
Results: The total effect of sex/gender on CHD mortality decreased with age. While the age-adjusted hazard ratio (men versus women) was 4.7 (95% CI: 3.5 to 6.1) in individuals younger than 50 years, it was only 1.9 (95% CI: 1.7 to 2.1) in the ≥50 years age group.
In the <50 years age group, the four major cardiovascular risk factors were able to explain 40.9% of this difference. The strongest factor was systolic blood pressure explaining 21.7% of the total sex/gender effect.
In the ≥50 years age group, the contribution of the risk factors was small amounting to only 8.2%. Single risk factors contributed less than 5%, with total cholesterol even showing a significant "negative" effect, i.e. mediation in favour of men.
Conclusions: The extent to which risk factors contribute to the gap between men and women regarding CHD mortality decreases strongly with age. Over the ages of 50 years, the persisting survival advantage of women can be explained only in small part through the pathways of major risk factors.

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Quitting smoke 'hits a late break' in acceleration of vascular aging

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Purpose: Vascular aging, as assessed by structural and functional properties of the arteries, is an independent indicator of cardiovascular risk. Smoking has a detrimental effect on arterial properties. We sought to investigate the effect of quitting smoke on the progression of vascular aging.
Methods: One hundred and forty-two subjects (mean age 51.9±10.8 years, 94 men, 61 hypertensives) with no established cardiovascular disease were investigated in two examinations over a 2-year period (mean follow-up visit 1.84 years). Subjects were categorized in current smokers, non-smokers and ex-smokers. Ex-smokers were further categorized according to the time elapsed since smoking (<5 years, 5–15 years and >15 years). Subjects had at the beginning and end of the study determinations of carotid-femoral pulse wave velocity (PWV). Based on these measurements the annual absolute changes were calculated.
Results: Smoking at baseline was not associated with statistically significant differences in PWV. However, the annual change was statistically different between the groups of smokers, non-smokers and the 3 groups of ex-smokers (p=0.041) after adjustment for relevant confounders. Specifically, smokers had 0.23m/s/year (95% CI: 0.10 to 0.35), non-smokers 0.17m/s/year (95% CI: 0.08 to 0.25), quitters (<5 years) had 0.28m/s/year (95% CI: 0.07 to 0.49), quitters (5–15 years) had 0.35m/s/year (95% CI: 0.11 to 0.59) and quitters (>15 years) –0.07m/s/year (95% CI: –0.26 to 0.13).
Conclusions: Quitting smoke seems to slow down progression of vascular aging after many years probably in an effort to compensate for former deleterious changes of smoking.

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Beta-blocker therapy optimization in elderly patients with left ventricular systolic dysfunction

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Introduction: The elderly population with left ventricle systolic dysfunction (LVSD) has been underrepresented in clinical trials of beta-blockers (BB) and maybe this is the reason why theses drugs are used less commonly and in lower doses in this group of population. The objective of this study is to evaluate the importance of the optimization of the medical treatment with BB in elderly population with LVSD.
Methods: We included all patients (pts) ≥75 years old, with LVEF ≤35%, studied in our center between January 2008 and April 2012. Clinical variables of interest were collected and clinical follow-up was performed. In each pt was collected information about treatment with BB and the dose reached. With this data we created a variable that determined the percent dose of BB (BB%) compared to the target level established in clinical guidelines (50 mg/d for carvedilol and 10 mg/d for bisoprolol). To analyze the effect of BB% on mortality and cardiovascular events (death, hospitalization for heart failure or ventricular arrhythmia), we used a Cox model adjusting for confounding and interaction with relevant clinical variables. In addition, to show the survival curves, the variable %BB was categorized into 3 groups (not BB, doses <50% and ≥50% doses).
Results: 556 pts were included. The mean age was 81.9 years, mean LVEF was 28% and there 34% of women. 143 pts (25.7%) did not take BB, 268 (48.2%) took low doses BB and 145 (26.1%) achieved high doses. During follow 223 pts died (40.2%), 92 in the untreated group, 97 in the low dose and 34 at the high dose. After adjusting the Cox model with confounding and interaction variables, we found