agents clopidogrel and prasugrel. METHODS: We developed a decision model to evaluate the potential clinical benefits and harms for three strategies—clopidogrel, prasugrel, and genotype-guided therapy—used for the treatment of patients with acute coronary syndromes with planned percutaneous coronary intervention. Data were derived from published literature, including clinical trials, and publicly available sources. The lifetime incidence of clinical events was projected, and net health benefit assessed using quality-adjusted life-years. Sensitivity and scenario analyses were conducted to assess uncertainty in results. RESULTS: Compared to clopidogrel therapy, prasugrel therapy was significantly associated with an average incremental decrease in the lifetime risk of myocardial infarction, but also an average incremental increase in the risk of major bleeds and bleed deaths. The genotype-guided treatment strategy did not exhibit any statistically significant difference in the population-level risks and benefits when compared to the clodipogrel and prasugrel strategies. No significant differences in net benefits were demonstrated among treatment strategies. The nonvariant clopidogrel therapy population and the prasugrel therapy population had significantly favorable cardiovascular and life expectancy outcomes compared to the CYP2C19 reduced-function allele population undergoing clopidogrel therapy. CONCLUSIONS: The inherent benefit-harm tradeoff of antiplatelet agents could potentially be improved with the use of CYP2C19 genotype information in patients with acute coronary syndrome. Genotype-guided therapy leads to improved outcomes for reduced-function CYP2C19 individuals, however this approach does not result in significant differences for the aggregate acute coronary syndrome population when compared to the overall clopidogrel and prasugrel therapy strategies. Ongoing randomized controlled trials will be critical in further assessing the role of the CYP2C19 reduced-function allele when deciding optimal antiplatelet treatment regimens.

PCV24

A META-ANALYSIS OF Efficacy AND SAFETY OF DALTEPARIN IN THE PREVENTION AND TREATMENT OF VENOUS THROMBOEMBOLIC DISEASE (VTE)

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OBJECTIVES: The purpose of this study was to evaluate the relative efficacy and safety of dalteparin against anticoagulant therapies in patients with cancer, medical patients at risk of VTE, total hip replacement, and acute myocardial infarction (AMI).

METHODS: A meta-analysis was performed with randomized clinical trials (RCT) where anticoagulant therapies were used to prevent or treat VTE. Effectiveness was assessed with the reduction in pulmonary thromboembolism (PE) and deep vein thrombosis (DVT) events; safety with the frequency and type of adverse events (AE). RCTs were searched in December 2008 in Medline, EMBASE and the Cochrane Collaboration. Two independent reviewers identified the abstracts, selected the full articles and extracted data. Odds ratios and weighted means differences were calculated. Random effects models were employed in the analyses.

RESULTS: From 2,439 abstracts, we obtained 91 RCT, 23 were excluded (unacceptable designs, insufficient outcome data) leaving 68. Dalteparin (2500–7500 IU/day) was compared against unfractionated heparin, enoxaparin, warfarin, nadroparin, fondaparinux, aspirin and placebo. In cancer patients, dalteparin showed to be effective in diminishing new infections and death (OR 0.86; 95% CI 0.73–0.99) or in worsening of QoL (OR 1.0; 95% CI 0.87–1.17). In comparison to placebo, the number of deaths were lower (OR 0.14; 0.02–1.27). In patients with VTE no statistical differences were found against competing alternatives, as well as in thromboembolism, thrombosis progression and death. Finally, in cancer patients, dalteparin showed to be effective in diminishing DVT (OR 0.47; 0.38–0.60) but not differences in reducing mortality (OR 0.92; 0.73–1.17; major bleeding (OR 1.20; 0.48–3.98) or minor bleeding (OR 0.87; 0.41–1.83). CONCLUSIONS: Dalteparin is an effective low-molecular-weight heparin in the prevention and treatment of VTE in surgery and non surgery patients, not showing higher AE than unfractionated heparin or other recommended therapies.

PCV25

PERCUTANEOUS CORONARY INTERVENTION COMPARED WITH AORTOCoronary BYPASS SURGERY IN DIABETIC PATIENTS WITH MULTIVESSEL CORONARY DISEASE

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OBJECTIVES: Diabetes patients with coronary artery disease represent a population with high cardiovascular morbidity and mortality. The objective of the study was to compare the long-term effectiveness of percutaneous coronary intervention (PCI) versus coronary artery bypass graft (CABG) in diabetic patients with multi-vessel coronary artery disease (MVD).

METHODS: Studies were retrieved from PUBMED database using keywords: angioscopy, coronary, stent, PCI and coronary artery bypass surgery (August 1992 to December 2009). Randomised controlled trials which compared PCI and CABG in head to head comparisons were included according to pre-specified inclusion/exclusion criteria. The outcomes of interest were mortality, myocardial infarction. Two reviewers independently extracted data from the included studies. Data was analyzed using STATA (v9.0). RESULTS: Of the 416 studies identified, 5 studies met the inclusion criteria. A total of 813 patients were included in this analysis (208 in ARTS, 78 in EARCI-II, 115 in MASS-II, 353 in BARI trial, and 59 in EAST). In total, 409 diabetic patients with MVD were randomized to PCI, and 404 were randomized to CABG. Survival was significantly greater after CABG than after PCI with a risk ratio of 1.28 (95% CI 1.06, 1.55); p = 0.009 for the five-year mortality rate. The ARTS trial was the only one that was randomized. Our objective was to determine what proportion of this decline was associated with temporal trends in CHD risk factors and advancements in medical treatments. METHODS: The validated IMPACT model was used for all analyses, integrating data on population size, CHD mortality, in addition to risk factor and treatment uptake changes in adults 25 years and older between 1994 and 2009 in Ontario. Relative risks and regression coefficients from the published literature quantified the relationship between CHD mortality and a) evidence-based therapies in 8 distinct CHD sub-populations (acute myocardial infarction (AMI), acute coronary syndromes, secondary prevention post-AMI, chronic angina/CHD, in-hospital, heart failure, community heart failure, and 1st prevention for hyperlipidemia or hypertension) and b) population trends in 6 risk factors (smoking, diabetes mellitus, systolic blood pressure, plasma cholesterol, exercise, and obesity). The outcome of interest was the cumulative survival. RESULTS: From 1994 to 2009, the adjusted CHD mortality rate in Ontario fell 35% from 190.9 to 124.8 deaths per 100,000 inhabitants, translating to an estimated 7585 fewer CHD deaths in 2005. Improvements in medical treatments accounted for approximately 45% of the total mortality decrease, most notably in AMI (9%), chronic angina (17%) and community heart failure (10%). Trends in risk factors explained approximately 48% of the total mortality decrease, specifically reductions in plasma cholesterol (23%), and systolic blood pressure (20%). Increasing diabetes prevalence and body mass index had a negative role by approximately 8%.

CONCLUSIONS: Our results suggest that future CHD strategies should maximise evidence-based therapies and support more aggressive policies to promote healthy diets.