French Society of Cardiology) have set up a consensus statement on the care of the hyperglycaemic/diabetic patient during and in the immediate follow-up of acute coronary syndrome (ACS). In particular, it includes the different phases of ACS [the intensive care unit (ICU) period, the post-ICU period and the short-term follow-up period after discharge, including cardiac rehabilitation] and also embraces all of the various diagnostic and therapeutic issues with a view to optimizing the collaboration between cardiologists and diabetologists. As far as diagnostic is concerned, subjects with HbA1c greater or equal to 6.5% on admission may be considered diabetic while, in those with no known diabetes and HbA1c less than 6.5%, it is recommended that an OGTT be performed 7 to 28 days after ACS. During hospitalization in the ICU, continuous insulin treatment should be initiated in all patients when admission blood glucose levels are greater or equal to 180 mg/dL (10.0 mmol/L) and, in those with previously known diabetes, when preprandial glucose levels are greater or equal to 140 mg/dL (7.77 mmol/L) during follow-up. The recommended blood glucose target is 140–180 mg/dL (7.7–10.0 mmol/L) for most patients. Following the ICU period, insulin treatment is not mandatory for every patient, and other antidiabetic treatments may be considered, with the choice of optimal treatment depending on the metabolic profile of the patient. Patients should be referred to a diabetologist before discharge from hospital in cases of unknown diabetes diagnosed during ACS hospitalization, of HbA1c greater or equal to 8% at the time of admission, or newly introduced insulin therapy or severe/repeated hypoglycaemia. Referral to a diabetologist after hospital discharge is recommended if diabetes is diagnosed by the OGTT, or during cardiac rehabilitation in cases of uncontrolled diabetes (HbA1c ≥ 8%) or severe/repeated hypoglycaemia.

**ISP8: Targeting normoglycemias in dysglycaemia patients at high CV risks: what are the benefits and risks?**

L. Rydén, Karolinska Institute, Stockholm, Dep Cardiology Solna, Karolinska University Hospital, Stockholm, Sweden

Impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and diabetes arise due to insufficient insulin secretion, and are risk factors for cardiovascular (CV) events. It has been argued that targeting normal fasting glucose levels with insulin may reduce CV events. This important question was studied in ORIGIN (Outcome Reduction with an Initial Glargine Intervention; ClinicalTrials.gov number, NCT00069784).

In ORIGIN people (n= 12537; mean age 63.5) with cardiovascular risk factors plus impaired fasting glucose, impaired glucose tolerance or type 2 diabetes were randomised to insulin glargine targeting a FPG <5.3 mmol/l (95 mg/dL) or standard glycaemic care for 6.2 (Inter Quartile Range 5.8, 6.7) years within a 2 x 2 factorial design. The two co-primary outcomes were CV death and non-fatal myocardial infarction stroke, and these events plus revascularization or hospitalization for heart failure.

Insulin glargine had a neutral effect on CV outcomes. The lecture will cover the design and outcome of the insulin part of the ORIGIN trial and put these results into perspective.

Further information may be derived from the following publications


**ISP9: Not all fat is bad in cardiovascular risk. Mechanistic considerations about visceral versus femoral adipose tissues.**

M. Lafontan, Inserm/UPS UMR 1048 - I2MC- Institut des Maladies Métaboliques et Cardiovasculaires, Toulouse, France

Fat distribution is a major determinant of health. Both sex and anatomic site differences in regional fat storage have been described. Gender-related and depot-specific differences exist in the expansion of the adipose tissue (AT) mass. Visceral obesity, which is easily measured by the expansion of waist circumference, includes abdominal subcutaneous and visceral adipose tissue (vAT). Accumulation of vAT increases cardiovascular disease and type 2 diabetes risks whereas expansion of subcutaneous AT (sAT) seems to protect from metabolic related diseases. These epidemiological observations have raised a number of questions that will be discussed in the talk. Why do obese individuals with upper body fat distribution have more health complications compared with obese individuals with lower body fat distribution? Why does the accumulation of vAT exert stronger deleterious effects than subcutaneous AT accumulation? Is vAT expansion a causal factor or only a marker of an altered metabolic status? Some mechanistic points will be considered in the talk. Adipogenic and angiogenic differences exist between fat depots. Differences in triglyceride synthesis have also been reported. Lipoprotein lipase, glucose and fatty acid uptake differ between visceral and subcutaneous AT. Lipolytic activity of adipocytes and catecholamine-induced mobilization of triglycerides differ with fat distribution. The clear depot-specific differences in the adrenergic response of the adipocytes in vitro, related to fat cell adrenergic receptors distribution and fat cell size, have been confirmed in vivo. Using in situ microdialysis, adrenergic stimulation of abdominal lipolysis appeared to be higher than femoral lipolysis. A technique based on arteriovenous difference sampling which allows measure of fatty acid trafficking across abdominal and femoral AT has revealed a lower catecholamine-dependent lipolytic rate in femoral AT than in the abdominal AT. Once stored in femoral AT fatty acids are not readily released. Entrapment of fatty acids would prevent their ectopic deposition in liver, muscle and pancreas. Noticeable differences also exist in production of hormones, cytokines and pro-inflamatory molecules in visceral and subcutaneous AT. Finally, multiple inflammatory cells infiltrate AT and accumulate in fat deposits of the obese. They perturb adipocyte biology and could contribute to the low grade inflammation in the obese. Macrophages number is proportional to the amount of body fat and macrophages are more abundant in vAT than in subcutaneous AT. To conclude body fat distribution has a major influence on risk factors. Delineation of new properties of “good fat pads” versus “bad fat pads” and their putative targeted manipulation offer promising perspectives.

**ISP10: A model for predicting cardiovascular risk in patients with type 2 diabetes mellitus based on the ADVANCE study**

F. Travert, Bichat Hospital and Paris 7 University, Paris, France

We developed a new model for predicting cardiovascular risk in people with type 2 diabetes mellitus based on the ADVANCE study since we considered there is a continuing need to develop new equations to estimate reliably cardiovascular disease. We used our 4.5-year follow-up cohort of the Action in Diabetes and Vascular disease: preterax and diamicron-MR controlled evaluation (ADVANCE) to estimate coefficients for significant predictors of CVD using Cox