clinical events (odds ratio [OR] 4.55, $P=0.037$ and OR 8.05, $P=0.002$, respectively). Analysis of RV variables only found that RV ejection fraction <45% was an independent predictor of major adverse clinical events (OR 5.60; $P=0.011$).

The authors conclude that ventricular dysfunction and severe RV dilatation are independent predictors of late major clinical events in adults with repaired TOF.

Original article Knauth AL *et al.* (2008) Ventricular size and function assessed by cardiac MRI predict major adverse clinical outcomes late after tetralogy of Fallot repair. *Heart* **94:** 211–216

Polymorphism in *KIF6* associated with coronary events in two independent cohorts

A large number of genetic polymorphisms have been linked with cardiovascular disease; however, few of these associations have been validated in independent studies. In a recent investigation, Iakoubova *et al.* conducted genetic association analyses to determine whether any of 35 polymorphisms previously shown to have an association with cardiovascular disease were associated with myocardial infarction (MI) in the placebo arm of the CARE trial, or with coronary heart disease (CHD) in the placebo arm of the WOSCOPS trial.

The researchers identified four single-nucleotide polymorphisms that were associated with MI in the CARE population and two single-nucleotide polymorphisms that were associated with CHD in the WOSCOPS population. Once the evidence from the two trials was combined and a multiple testing correction was applied, however, only a Trp719Arg polymorphism in the gene that encodes kinesin-like protein 6 (*KIF6*) remained significantly associated with coronary events. In the placebo arm of the CARE trial, carriers of this polymorphism had an increased risk of recurrent MI even after adjustment for age, sex, smoking history, BMI, cholesterol level, hypertension, and diabetes mellitus (hazard ratio 1.50, 95% CI 1.05–2.15). The risk of CHD in carriers of the *KIF6* 719Arg allele in the placebo arm of the WOSCOPS trial was similarly increased after adjustment for conventional risk factors (odds ratio 1.55, 95% CI 1.14–2.09). Further analyses revealed that pravastatin treatment markedly lowered the risk of coronary events in carriers of the *KIF6* 719Arg allele in both trials.

Original article Iakoubova OA *et al.* (2008) Association of the Trp719Arg polymorphism in kinesin-like protein 6 with myocardial infarction and coronary heart disease in 2 prospective trials: the CARE and WOSCOPS trials. *J Am Coll Cardiol* **51:** 435–443

Impaired perfusion after MI is associated with ventricular tachycardia and fibrillation

Poor myocardial perfusion after myocardial infarction (MI) is associated with scar development and impaired left ventricular function, and might, therefore, lead to an increased risk of sustained ventricular tachycardia (VT) and/or ventricular fibrillation (VF) and subsequent sudden cardiac death. To assess this relationship, Gibson *et al.* retrospectively analyzed the coronary angiograms of patients with ST-segment elevation MI enrolled in the CLARITY-TIMI 28 trial who had recorded VT and/or VF.

Patients with impaired perfusion (thrombolysis in myocardial infarction [TIMI] perfusion grade 0–2) had a higher incidence of VT and/or VF events than those with normal perfusion (TIMI perfusion grade 3; 7.1% vs 2.6%, $P<0.001$). The propensity for VT and/or VF remained in patients with TIMI flow grade 3 or preserved systolic function—those with impaired perfusion still had a raised incidence of VT and/or VF events, despite having normal epicardial flow (5.4% vs 2.8%; $P=0.01$) or a left ventricular ejection fraction ≥30% at angiography (4.7% vs 2.7%; $P=0.04$). Of the patients who experienced VT and/or VF events at any point during hospitalization, 30-day mortality was higher in those with impaired perfusion than in those with normal perfusion (17.5% vs 2.4%; $P=0.02$).

The authors conclude that in patients with ST-segment elevation MI, including those with normal epicardial flow or systolic function, impaired myocardial perfusion is associated with an increased risk of VT and/or VF events and of mortality.

Original article Gibson CM *et al.* (2008) Association of impaired thrombolysis in myocardial infarction myocardial perfusion grade with ventricular tachycardia and ventricular fibrillation following fibrinolytic therapy for ST-segment elevation myocardial infarction. *J Am Coll Cardiol* **51:** 546–551