**PLATELET FUNCTION AND CLINICAL ISCHEMIC OUTCOMES**

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Acute coronary syndrome (ACS) results from formation of a thrombus on a ruptured coronary artery plaque. Primary treatment of ACS includes anti-platelet agents, such as aspirin, clopidogrel, or newer P2Y12 ADP inhibitors, such as prasugrel or ticagrelor. Patients who are resistant to anti-platelet therapy have a worse outcome, So platelet function testing may be helpful in assessing risk. Genetic variants have also been associated with greater risk in ACS. Cytochrome P450 2C19\*2 genotype and other P450 variants (causing reduced conversion of clopidogrel from the pro-drug to the active drug) are associated with lesser clopidogrel effect and worse outcomes following coronary intervention. Other P450 variants, such as 2C19\*17 produce increased conversion to the active drug and greater clopidogrel effect. The newer P2Y12 inhibitor prasugrel, which depends less on 2C19 for conversion to active drug, may be more effective, particularly in patients with loss of function P450 variants, but may also be associated with more bleeding and other side effects, and is much more expensive. The issue of 2C19 variants may be more important in ACS patients treated with PCI and coronary artery stenting, where platelet-induced stent thrombosis is a major concern. However, two recent large meta-analyses do not support the clinical usefulness of 2C19 genotyping, even for predicting stent thrombosis. Despite offering promise, the role of genetic testing in people with ACS or at risk for ACS needs further study.