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ANTIPLATELET THERAPY

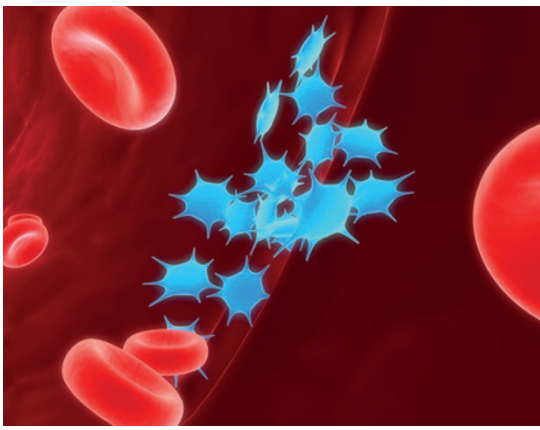
# ARCTIC leaves platelet testing out in the cold

ARCTIC and the TRILOGY ACS platelet function substudy, both presented as late-breaking clinical trials at the AHA 2012 Scientific Sessions, have called into question the utility of platelet-function testing. The efficacy of oral P2Y12 antagonists can now easily be monitored using commercially available platelet-function assays, such as the VerifyNow® P2Y12 assay (Accumetrics, Inc., San Diego, CA, USA). Whether a reduced level of platelet reactivity translates into a decreased risk of cardiovascular events is unclear.

The ARCTIC investigators compared conventional care with a strategy of platelet-function monitoring involving drug adjustment in patients who had a suboptimal response to antiplatelet therapy. The researchers enrolled 2,440 patients scheduled to undergo coronary stenting in 38 centres in France. In the monitoring group, 34.5% and 7.6% of patients taking clopidogrel or aspirin, respectively, had high platelet reactivity, and their drug regimen was adjusted accordingly during percutaneous coronary intervention. The composite primary end point (death, myocardial infarction, stent thrombosis, stroke, or urgent revascularization up to 1 year after stent implantation) occurred in 34.6% and 31.1% of patients in the platelet-testing and conventional-care groups, respectively (HR 1.13, 95% CI 0.98–1.29). The rate of major bleeding did not differ significantly between the groups, and the investigators concluded that the "study showed no significant improvements in clinical outcomes with platelet-function monitoring and treatment adjustment for coronary stenting".

In the TRILOGY ACS platelet function substudy, 27.5% ($n = 2,564$) of the overall TRILOGY ACS study population underwent regular platelet-reactivity testing. Enrolled patients had medically managed unstable angina or non-ST-segment elevation myocardial infarction and were randomly allocated to receive either clopidogrel or prasugrel. Overall, prasugrel was associated with a lower level of on-treatment platelet reactivity than clopidogrel. However, no significant relationship existed between the rate of the primary composite end point (cardiovascular death, myocardial infarction, or stroke) and the continuous platelet-reactivity level during 30-month follow-up. Dr Matthew Roe, lead author of the trial report, suggests that these findings "might explain the comparable clinical outcomes observed with prasugrel versus clopidogrel in the main TRILOGY ACS trial".

Dr Richard Becker from Duke University, Durham, NC, USA puts the data into context. "The findings from



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TRILOGY ACS show that routine platelet-function testing ... does not play a role in the routine management of patients with ACS being treated medically with second and third generation thienopyridine platelet antagonists. The findings, however, do not call into question the importance of platelets and platelet-related biological processes in the natural history of ACS and related phenotypes."

***Gregory B. Lim***

**Original articles** Collet, J.-P. *et al*. Bedside monitoring to adjust antiplatelet therapy for coronary stenting. *N. Engl. J. Med.* doi:10.1056/NEJMoa1209979 | Gurbel, P. A. *et al.* Platelet function during extended prasugrel and clopidogrel therapy for patients with ACS treated without revascularization: the TRILOGY ACS platelet function substudy. *JAMA* **308**, 1785–1794 (2012)