New insights into platelet function testing: From bench to bedside

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The history of platelet function testing intercalates with anti-platelet drug development. Indeed, the original bleeding time test was developed to assess platelet disorders associated with bleeding symptoms. However, this test was found ineffective for testing the response to anti-platelet drugs.

The first anti-platelet drug, aspirin, was originally developed as an anti-inflammatory agent late in the 19th century. It was some 60 years later when this drug was adopted as an anti-platelet agent in the setting of cardiovascular diseases. This development promoted the invention of the first method and device for testing platelet function and their response to aspirin. In their original manuscript, Born et al. tested the response of platelets to ADP and applied it to monitor the response to platelet inhibitors (1, 2). Indeed, the original aggregometer is still considered a gold standard for platelet function testing as well as for the assessment of their inhibition.

The field of anti-platelet therapy is currently undergoing a dramatic revolution, with the introduction of many new agents for both acute intervention as well as for the secondary prevention of recurrent coronary events. The introduction of new technologies of percutaneous coronary intervention (PCI), was associated with early and late complications, including stent thrombosis, which led to intensified protocols of anti-platelet therapy. Recently a diverse response to anti-platelet therapy has been observed and a link between this response and clinical outcome suggested. These developments triggered the search for a tool of testing the response to anti-platelet therapy in an effort to improve clinical outcome. Many clinical studies have tested the response to aspirin, clopidogrel and the new agents, applying different testing systems and have tried to correlate it to clinical outcome.

The current Theme Issue in this journal on “Platelet function testing – from bench to bedside”, provides review articles as well as some original contributions, all of which are dedicated to the question of whether specific platelet function testing can offer a personalised dose adjustment, which will lead to improved clinical outcome.

In their review, Sibbing and colleagues are setting the stage by defining the term of high platelet reactivity including underlying mechanisms, describing the different methods of its identification, and potential future directions (3). Fefer et al. provide a review in greater detail on the genetic basis of platelet responsiveness to clopidogrel (4). Additional clinical and confounding factors affecting the individual response to clopidogrel are discussed in another overview by Gremmel and co authors (5). A sub-study of the TIMI–44 is reported by Frelinger et al., who describe a correlation between platelet reactivity before therapy, and the response to clopidogrel (6). Two additional reports are dealing with the question of which method is suitable for testing the response to anti-platelet drugs; thus Voisin et al. compare the results obtained by the VASP assay to those of the VerifyNow test (7), whereas Freynhofer and colleagues compare the VASP to the multiple electrode aggregometry test (8).

The association of the response to aspirin among patients presenting with cerebrovascular ischaemia is reported by Law et al. (9), whilst Gurbel and colleagues address the issue of peri-operative platelet function testing and its potential effect on clinical outcomes (10). Finally, Angiolillo and co-workers report on the adjunctive cilostazol therapy on platelet function profiles in patients with and without diabetes mellitus on aspirin and clopidogrel therapy (11), whilst Gurbel et al. discuss some implications of the recently reported GRAVITAS study (12).

This Theme Issue continues the long standing interests of this journal in platelet function testing (13–20). Previously published work has ranged from novel ELISAs for platelet function testing (13) to assessment of various pathophysiological insights into platelet function per se (14–18). Of course, aspirin dose can influence platelet physiology (19), but ultimately, platelet (dys)function is associated with bleeding (20).

As Guest Editors, we sincerely hope you will enjoy and find this Theme Issue a useful addition and contribution to the field.

Conflict of interest
None declared.

References
5. Gremmel T, Panzer S. Clinical, genetic and confounding factors determine the dynamics of the in