A WORD FROM THE EDITORS:
First, we would like to thank Dr Marc Fisher for asking us to be the editors of this section, and Drs Geoffrey Donnan and Stephen Davis for their contribution over the past several years. Second, we encourage the readers to contact us directly with their feedback or suggestions for future topics.

The Case:
A patient presents with a recurrent lacunar infarct. He was already taking a baby aspirin.

The Questions:
(1) Should the dose of aspirin be increased to 325 mg or should aspirin be changed to another antiplatelet agent?
(2) Should platelet function assays be used to guide treatment?

The Controversy:
THE USE OF PLATELET FUNCTION ASSAYS MAY HELP TO DETERMINE APPROPRIATE ANTIPLATELET TREATMENT OPTIONS IN A PATIENT WITH RECURRENT STROKE ON A BABY ASPIRIN.

Platelet Function Assays in Stroke Management
More Study Is Needed

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Antiplatelets, especially aspirin, remain the main cornerstone therapy for ischemic stroke prevention. The question always arises in everyday practice of what to do when a patient has a recurrent stroke at the time of already being on a baby aspirin. Some advocate increasing the dose of aspirin; others recommend substituting it with another antiplatelet agent such as clopidogrel or the combination of extended-release dipyridamole and aspirin. As indicated by the eloquent comments made by our protagonists, higher doses of aspirin are associated with increased risk of side effects, including bleeding complications, and their added benefit in preventing stroke recurrence is debatable or not very compelling.1-3 In practice, poorer adherence to higher doses of aspirin resulting from intolerance or side effects may counterbalance any potential greater efficacy. Therefore, we would generally switch patients who have a recurrent stroke at the time of taking a baby aspirin to another antiplatelet agent.

It is important to keep in mind that stroke recurrence at the time of taking aspirin does not always equate with aspirin “failure.” Drs Eikelboom, Emery, and Hankey remind us of the potential effects of drug interactions such as concomitant use of nonsteroidal anti-inflammatory drugs, which are often overlooked or minimized when dealing with patients with stroke. Poor compliance may be a relevant issue in certain stroke populations commonly underrepresented in randomized controlled trials. To look at the big picture, although antiplatelets are effective in reducing stroke recurrence, their benefit is modest. Optimizing the management of poorly controlled risk factors such as blood pressure, diabetes, smoking, and hyperlipidemia may be all that is required in aspirin-treated patients who experience a recurrent stroke. The mechanism of the recurrent stroke is also important. Altering the dose of aspirin or substituting it with another antiplatelet agent is unlikely to be effective if the recurrent stroke is cardioembolic in nature, in which anticoagulation is likely to be more effective.

Dr Alberts offers helpful insights into the issue of aspirin resistance and raises the potential role of combined therapy with aspirin and clopidogrel to overcome aspirin resistance. We urge caution in using this combination in patients with stroke unless indicated from a cardiovascular standpoint or in
patients with stents based on the results of the Management of Atherothrombosis With Clopidogrel in High-Risk Patients (MATCH) trial.4

Our authors highlight the promise and limitations of the currently available platelet function assays. We agree that the concept of performing a test to know if a medication is working or not working as intended is logical, reasonable, and appealing; however, the question remains as to whether the use of platelet function assays or other genetic tests to dictate the choice of the antiplatelet agent is ready for prime time and widespread implementation. We fear that the answer is no, not at this time; more study is needed.

Let us look at the big picture again. First, the currently available assays are not standardized and different tests can produce different results. Second, drug resistance is not an all-or-none phenomenon and the results of these tests are not uncommonly intermediate and not “black or white.” So, an optimal therapeutic range for antiaggregation response is unclear. Third, other alternative and more effective anti-platelet agents are available5,6 and testing for aspirin resistance is only likely to be necessary in circumstances in which the cost of these alternatives is prohibitive. Fourth, there are no data to suggest whether altering aspirin dose or antiplatelet therapy based on a platelet function test reduces stroke recurrence rate.

In the absence of such data and lack of consensus on what test to perform, we do not believe that platelet function assays are ready to be used in the routine management of patients with stroke who are taking aspirin. We agree that a consensus on a testing paradigm, normal ranges, and standardized procedures are needed to better define the potential use of these tests and recommend that they are used within the scope of a prospective clinical trial, a database, or a registry if they are to be used at this point in time.

Disclosures
None.

References

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