Dear Sir,

We have read with interest the paper by Pontiggia et al. (1), recently published in Thrombosis and Haemostasis (1). The authors reported that, under conditions of high shear rates, the release of microparticles from platelets was strongly reduced by adding anti-GPIb antibodies (1). We would like to comment on this study.

Microparticles are membrane vesicles shed by platelets after activation (2), and they carry antigens characteristic of intact platelets, such as functional adhesion receptors glycoprotein GPIb, GPIIb/IIIa, and P-selectin (3, 4). Microparticles also provide a disseminated catalytic surface of phosphatidylserine that accelerates coagulation (5), and are thought to be thrombogenic (4). Platelet microparticle membranes have 50- to 100-fold higher specific procoagulant activity than activated platelets (6). The authors reported that blocking GPIb by antibodies is more effective in suppressing microparticle formation and thrombin generation than blocking GPIIb/IIIa with inhibitors. The authors, however, have not excluded that there is an immediate effect of these monoclonal antibodies on the microparticles in their experimental set-up.

It is known that microparticles can bind to other cells, and it is quite possible that the antibodies influence the binding of microparticles to other blood elements such as leukocytes or monocytes (7, 8). Inhibition of the binding of microparticles to other blood elements should give increased levels of measured microparticles in a FACS set-up due to decreased binding to other cells. Contrary to expectation, according to the paper by Pontiggia et al. (1), an inhibition of microparticle release occurs when such antibodies have been added. We suggest the authors should consider introducing controls in which leukocytes or monocytes are depleted. Therefore, increased inhibition of microparticle release than that reported in the article by Pontiggia et al. may occur.

References