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Thromboelastograph (TEG) utilization for diagnosis and personalized treatment of complex coagulopathies

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This is a brief description of what is TEG: The new look at TEG; an old test first developed in 1948 and until recently used almost exclusively in open heart and liver transplant procedures came after a series of revelations about the coagulation process. Clinicians already recognized that the classic coagulation cascade model with intrinsic and extrinsic pathways leading to haemostasis though admittedly complicated did not fully describe all the factors at work in vivo. Potential TEG and its modifications (rTEG and TEG platelet mapping) use as a global assay of haemostasis in various clinical settings will be discussed during this talk.

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Contribution of the activated protein C system to the dynamics of thrombin formation by human vascular cells and derived microparticles

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Background & Objective: Endothelial (EC) and vascular smooth muscle cells (SMC) constitutively synthesize the tissue factor pathway inhibitor (TFPI) which cooperates in plasma with the activated protein C (APC) system to provide a synergistic inhibition of thrombin formation. We thus investigated the abilities of vascular cells to support thrombin formation and inhibition in comparison with platelets. In addition, we examined whether the surface of microparticles (MP) derived from these cells exhibit the same phenotype.

Methods: Thrombin generation and its limitation by APC were assessed by thrombography using as cellular surfaces either human platelets, cultured aortic SMC or EC or their MP generated in response to ionomycin activation. APC sensitivity was expressed as the APC concentration (IC₅₀-APC) needed to reduce the endogenous thrombin potential (ETP) by 50%.

Results: Compared with platelets, EC and SMC had a two-fold lower ETP in the absence of APC. TFPI as well as the endogenous APC generated at the surface of cells contributed to the lower ETP values. Sensitivity to exogenous APC was higher for EC and SMC than for platelets: IC₅₀-APC values were 15.1±0.9, 11.1±0.6 and 8.5±0.5 nM respectively. Significant differences were abolished by an anti-TFPI antibody. Thrombin generation with MP generated from CML or EC was 2-fold higher and sensitivity to APC 2-fold lower compared with parent cells. For platelets and platelet-derived MP, ETP values were similar and sensitivity to APC was paradoxically 3 to 4-fold higher with MP.

Conclusion: These findings demonstrate that the in vitro anticoagulant effect of APC amplified by a synergistic effect of TFPI is higher at the level of SMC than EC. The contribution of APC for MP derived from EC and SMC is less functional compared to their respective parent cells. The APC pathway may therefore represent a crucial target to modulate the thrombogenicity of vascular cells and derived MP after injury.

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