

Modifications de l'hémostase sous assistance circulatoire mécanique (ACM)

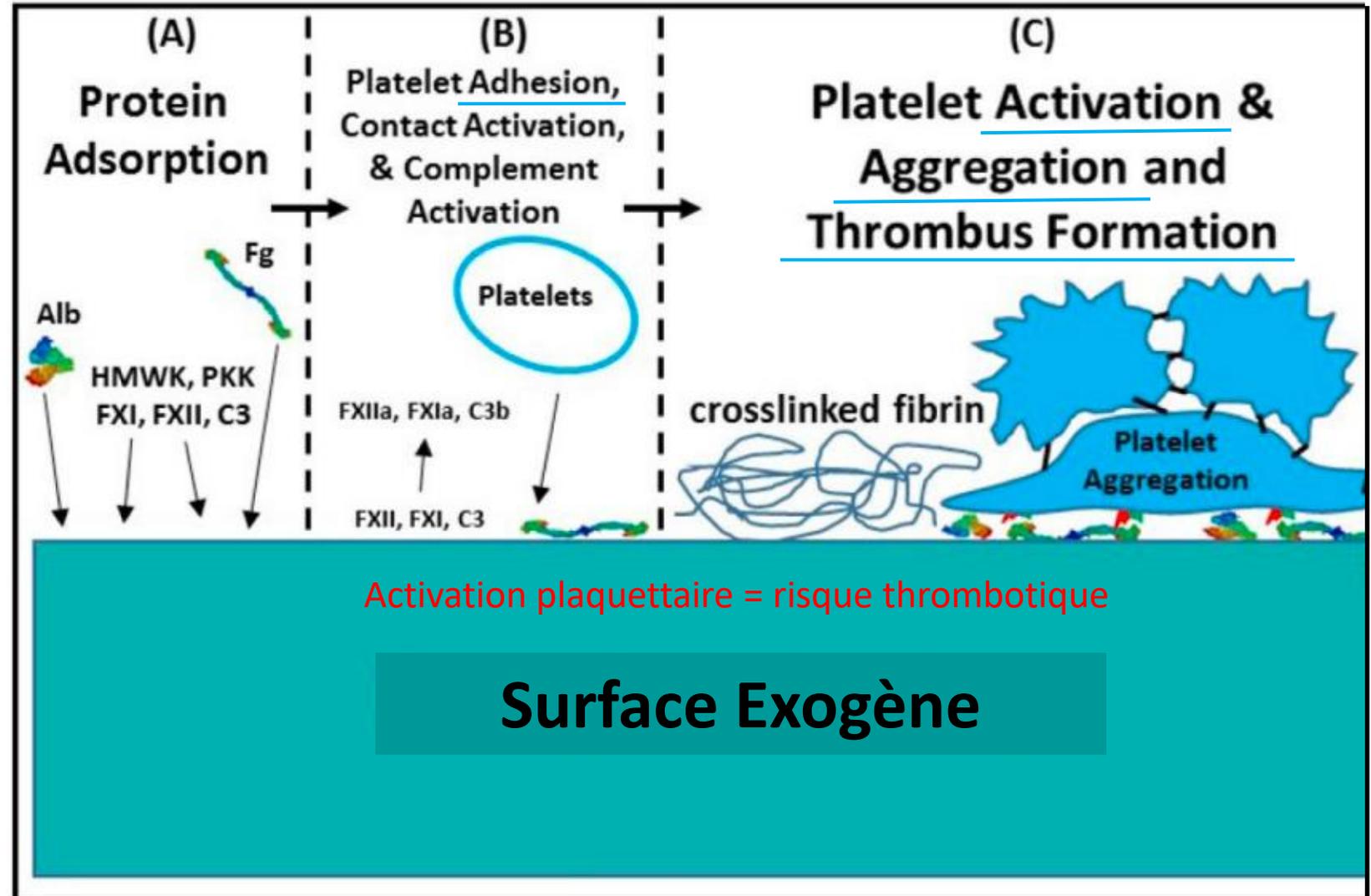
Conflit d'intérêt

- Aucun

Adsorption protéique

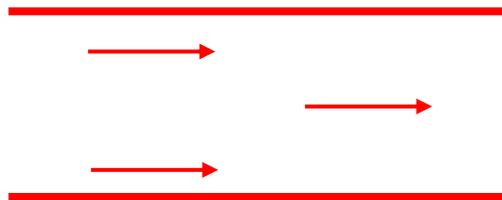
favorisée par les interactions électrostatiques et hydrophobes:

- Fibrinogen
- Fibronectin
- von Willebrand factor
- FXI-FXII
- Kininogen
- Prekallikrein



Contrainte de cisaillement (shear stress): forces frictionnelles exercées par le flux sanguin contre la paroi vasculaire, déterminé par le **débit sanguin**, la **viscosité du sang** et le **diamètre du vaisseau**

Vascular system



ACM

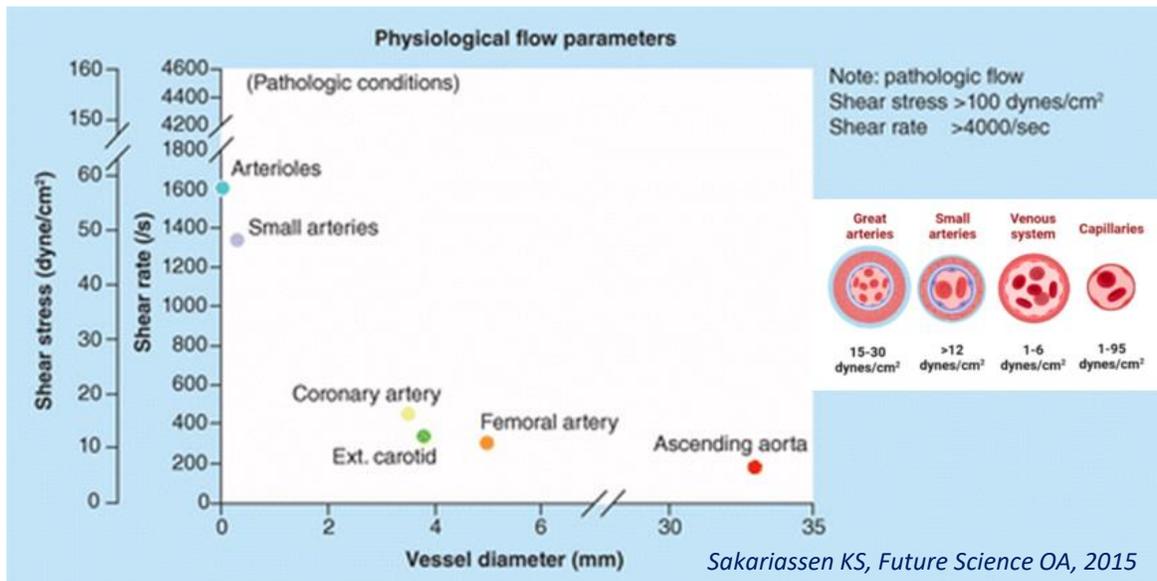
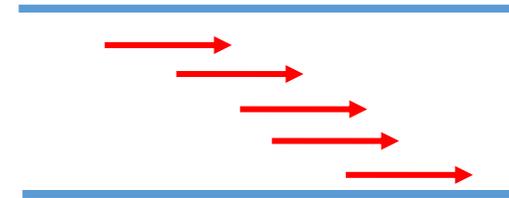
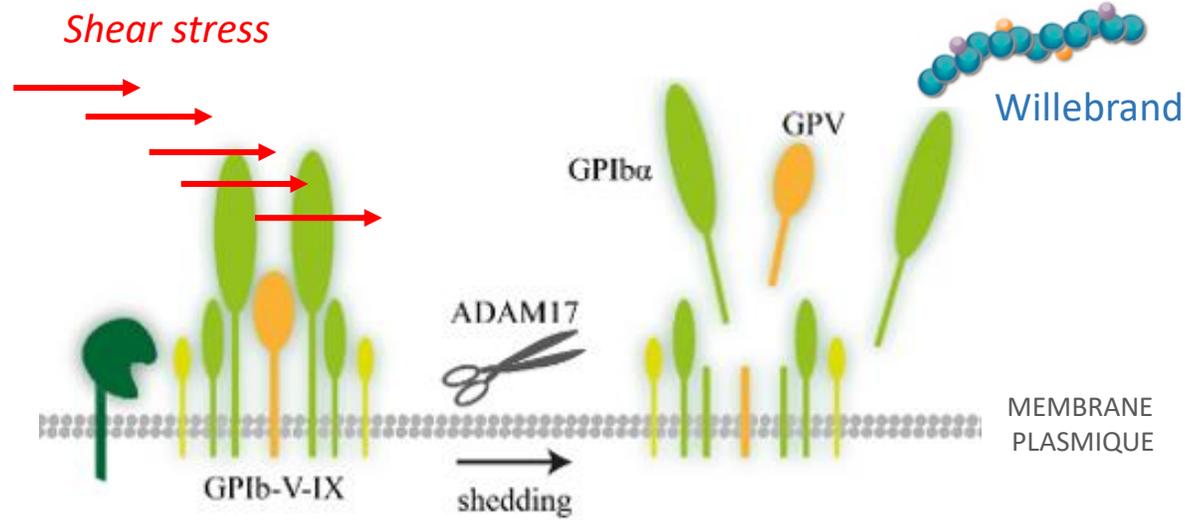


Table 1. Typical range of wall shear rates and stresses in blood vessels

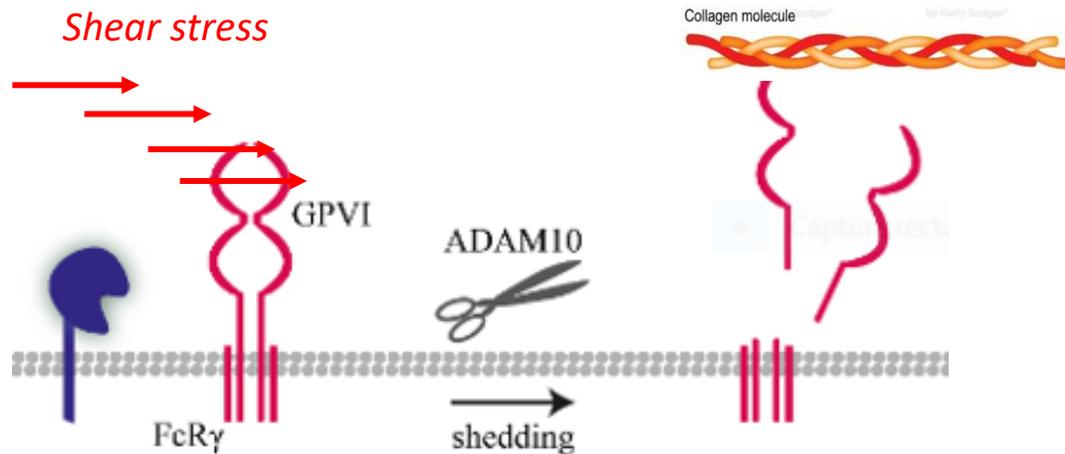
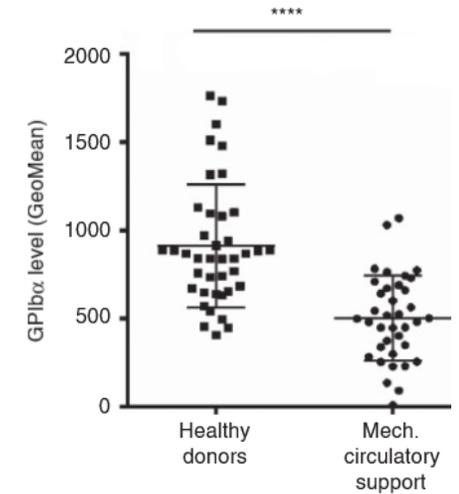
Blood vessel	Shear rate (/s)	Shear stress (Pa)
Large arteries*	300-800	1.4-3.6
Arterioles*	450-1600	2-7.2
Veins*	15-200	0.07-0.9
Stenotic vessels*	800-10 000	3.6-45
Axial flow LVAD†	—	600
Centrifugal flow LVAD†	—	150-230

X100
X20-30

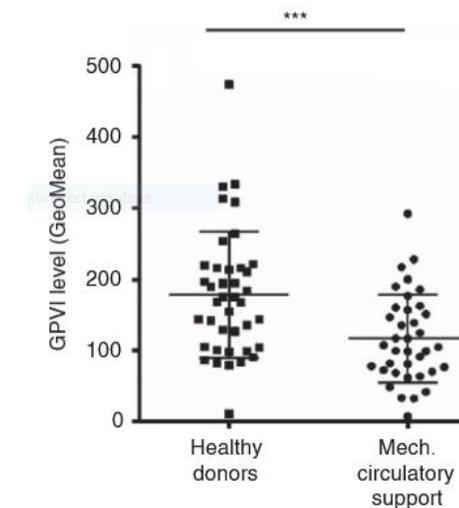
TAUX DE CISAILLEMENT ÉLEVÉS ET PERTE DES RÉCEPTEURS PLAQUETTAIRES



Récepteur
Willebrand

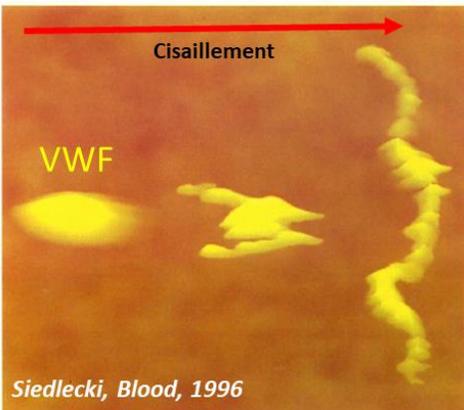


Récepteur
collagène



HÉMOSTASE, FACTEUR WILLEBRAND ET CONTRAINTES DE CISAILLEMENT (SHEAR STRESS)

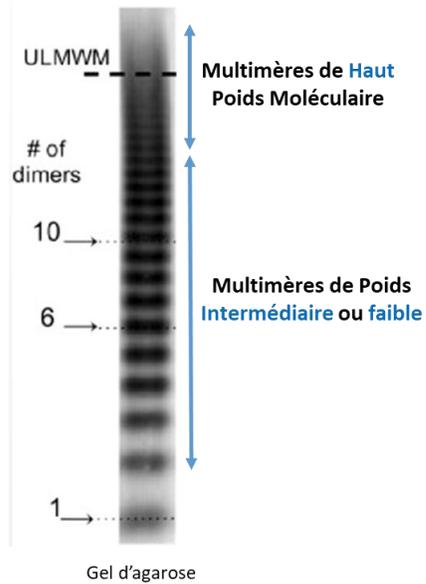
No Shear → Shear Applied by AFM Probe → 35 Dyn/cm² Applied by Rotating Disk



Globular vWF Short Extended Chain Extended Chain

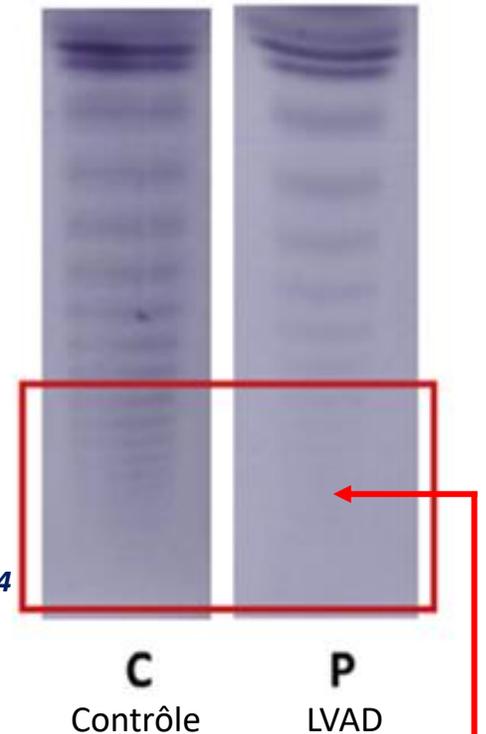
Shear-Dependent Changes in the Three-Dimensional Structure of Human von Willebrand Factor

Conformation native
Non fonctionnelle



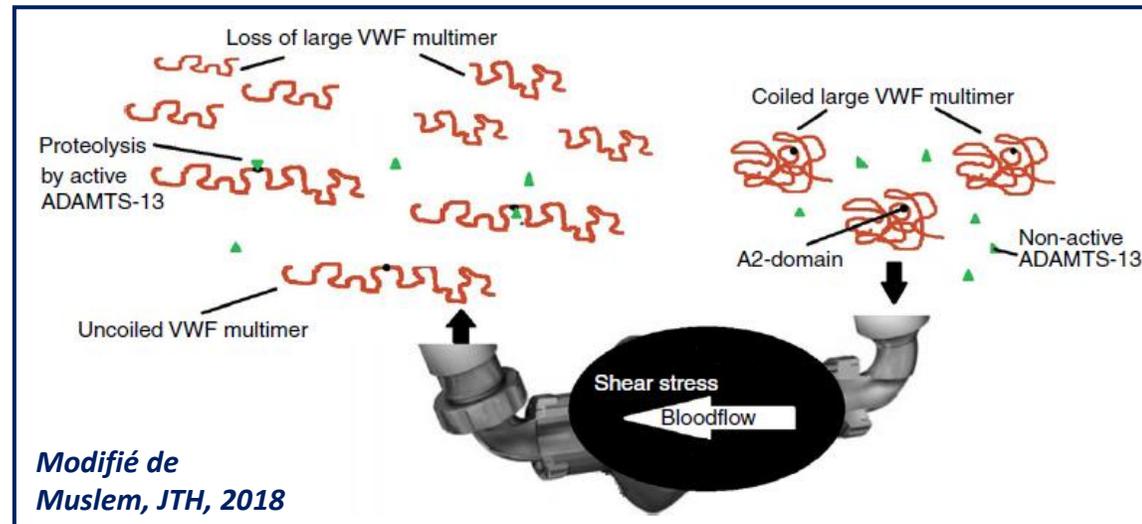
ACM

Fiore, JTCS, 2014



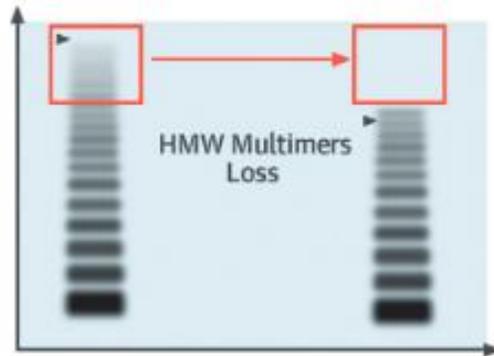
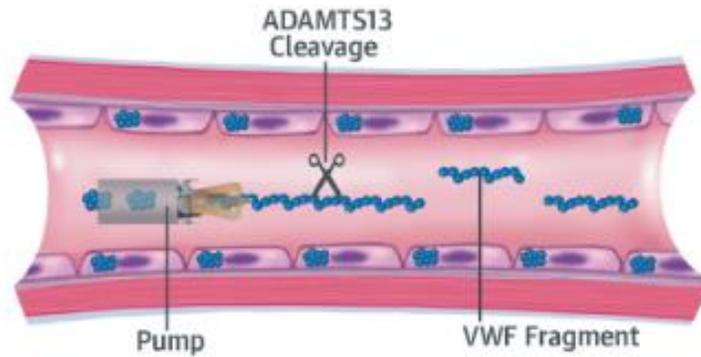
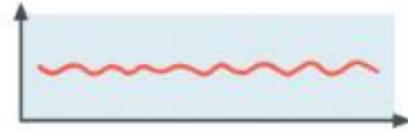
Perte des multimères de haut poids moléculaires et de fct du VWF

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Maladie de Willebrand Acquis
=
Risque hémorragique

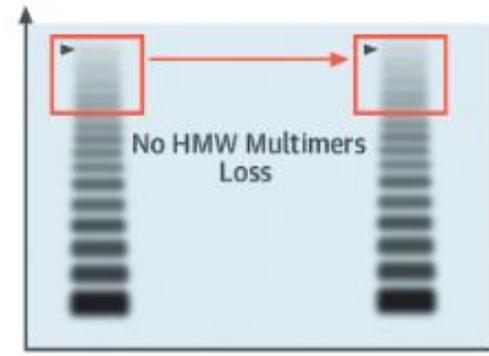
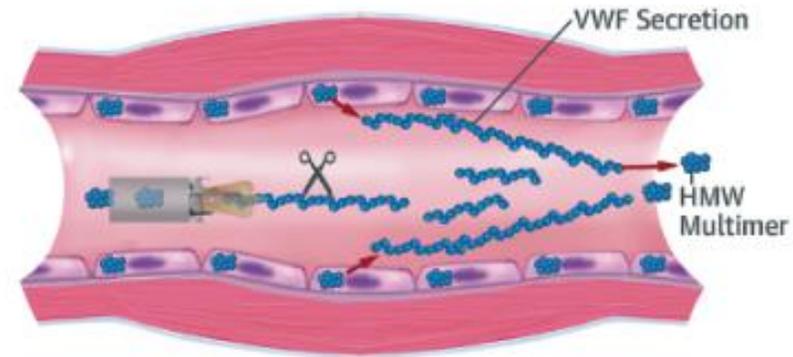


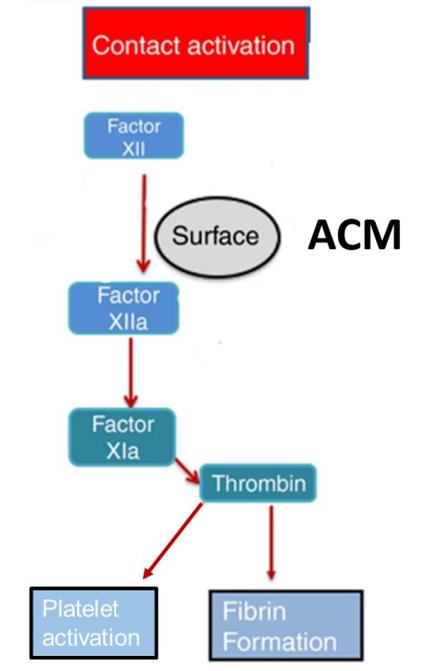
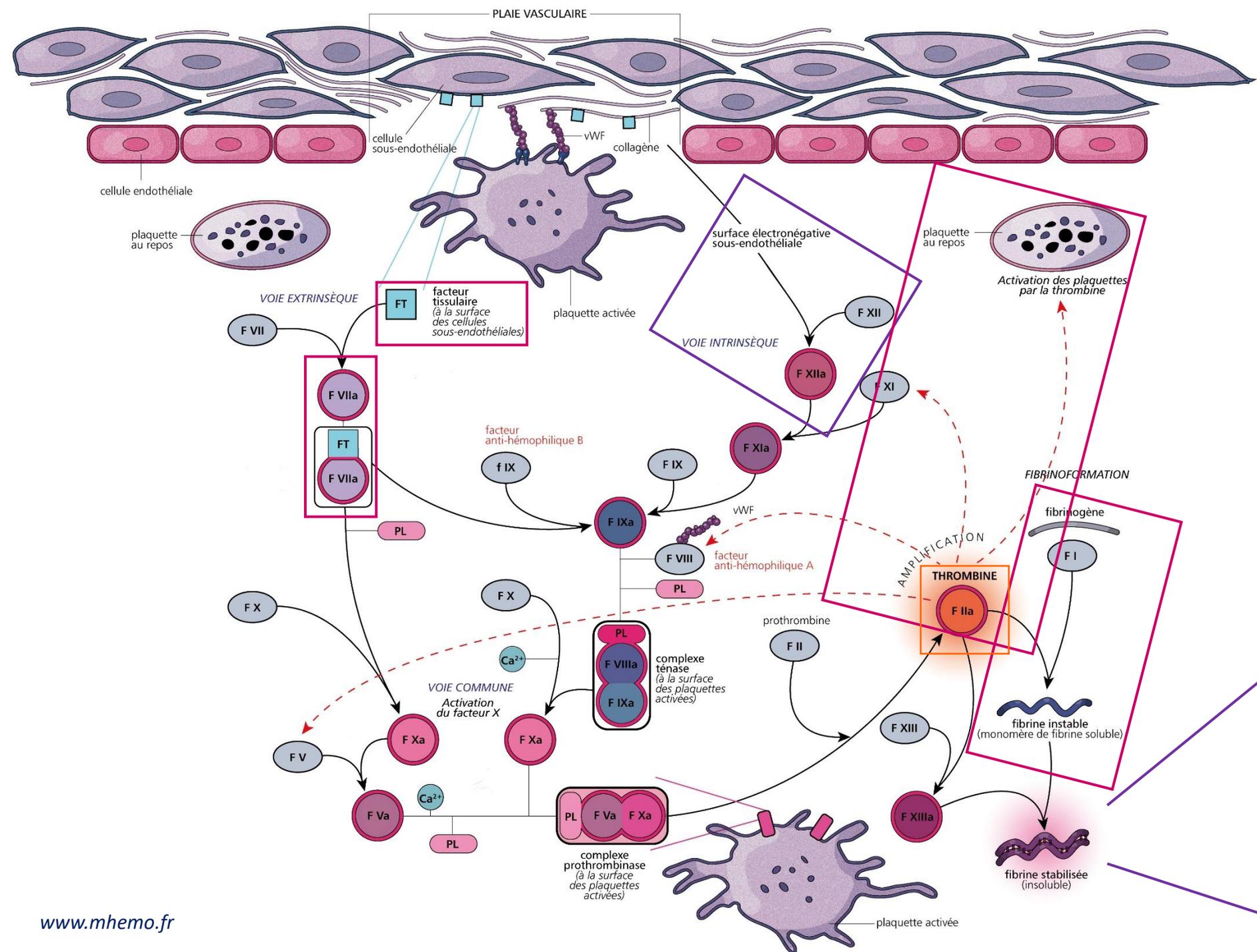
PULSATILITÉ ET WILLEBRAND ACQUIS

Low Pulsatility

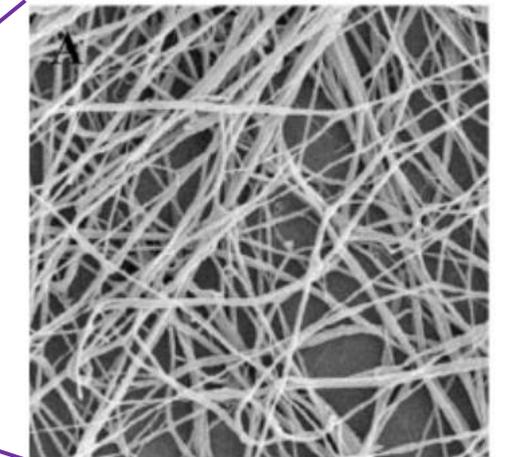


Normal Pulsatility





**HEMOSTASE:
GENERATION DE THROMBINE**



MODIFICATIONS DE L'HÉMOSTASE SOUS ACM

