Description for a problem studied at the UK Mathematics-in-Medicine Study Group Nottingham 2002
< https://mmsg.mathmos.net/uk/2002/plaque-rupture/ >

The role of neovessels in the development and progression of atheromatous disease.

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Vascular disease causes significant morbidity and mortality in the western world. Vessel wall disease includes a spectrum of conditions from benign fatty streaks in early life to complicated atheromatous plaques resulting in vessel narrowing and distal thromboembolism. It has been recently realised that the vessel wall is not as passive conduit of blood but rather a highly complex organ that interacts with its environment. The major function of the vessel wall, and more specifically the endothelial lining, is to maintain adequate blood flow to the end organ it is supplying. This is achieved by regulatory mechanisms that control vascular tone, inflammation and anticoagulation.

Just as with any organ the endothelium is prone to disease and is continually exposed to numerous potentially harmful processes, whether mechanical or chemical. The response of the endothelium to these will dictate development of disease and this in part will be dependant on the morphology and physiology of the vessel wall. The endothelium requires an adequate supply of nutrients to maintain healthy function and while some of these can be derived from the vessel lumen itself, in larger vessels a separate supply dedicated to the vessel wall is required (vasa vasorum). One response to vessel wall disease is the proliferation of these small blood vessels in an attempt to maintain satisfactory blood supply. Regrettably in so doing the vasa vassorum become liable to rupture causing bleeding within the atheromatous plaque. This has a number of effects: if the bleeding is sufficiently severe there will be a rapid enlargement of the plaque potentially causing occlusion of the vessel; a resultant breach of the plaque surface exposes the contents of the plaque to the systemic circulation triggering thrombosis and distal embolisation; less severe bleeding will lead to slower progression of the plaque enlargement but may also cause surface endothelial activation again making it prone to platelet aggregation and embolisation. All these will lead to a progression of disease and potential symptoms arising from the end-organ due to decreased blood flow or thromboembolism causing distal ischaemia and infarction.

Q1. Atheromatous disease commonly arises at the bifurcation of arteries, thought to be due to the effect of turbulence at this site and subsequent endothelial damage. The vasa vasorum flow is derived from upstream supply and therefore it could be hypothesised that for a short distance the vessels walls distal to the bifurcation are being supplied by the vessel proximal to the bifurcation. Does this change in morphology cause any local reduction in vasa vasorum flow/pressure as one vessel divides into two?

Q2. The vessel wall is dependant on two nutrient supplies – one from the lumen and one from the vasa vasorum. What nutrient/oxygen gradient exists across the vessel wall and what is the effect of disease (thickening) on this?

Q3. As the plaque slowly enlarges it will encroach more into the vessel lumen causing stenosis. At what degree of stenosis will there be a significant pressure gradient across the stenosis?

Q4. Atheromatous plaque causes vessel narrowing. The stenosis will eventually cause a significant pressure gradient. The vasa vasorum obtain their supply from upstream and therefore may be exposed to a higher pressure than the plaque which is exposed to the lumen adjacent to the distal part of the plaque. What effect will this have on the potential site of vasa vasorum rupture, haemorrhage and potential plaque rupture/activation assuming the idealised shape of the plaque is domed?