

# Adventitia Reinforcement Surgery to Treat Atherosclerotic Disease and Aneurysm

Xinggong Wang, PhD

Department of Cardiology, Renji Hospital Affiliated to Shanghai Jiaotong University School of

Medicine, Shanghai, China

Email: [xinggangwang11@fudan.edu.cn](mailto:xinggongwang11@fudan.edu.cn)

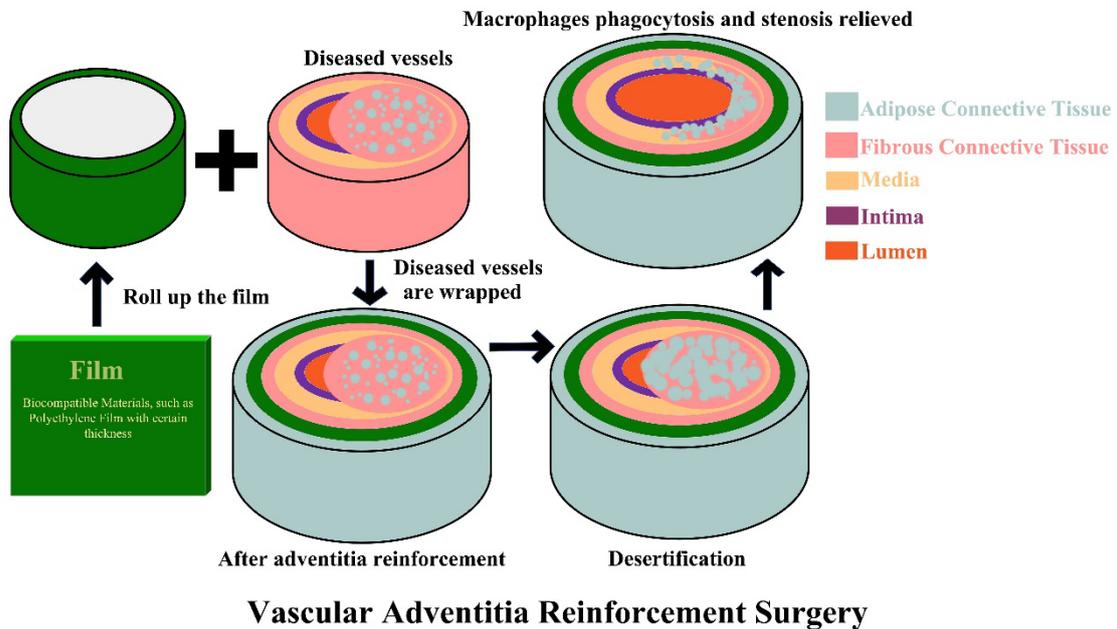


Figure 1. Vascular Adventitia Reinforcement Surgery to Treat Atherosclerotic Disease and Aneurysm

## Main text

**Atherosclerotic** disease and aneurysm are now the most common diseases threatening human health. Although there are many treatment methods, such as drugs therapy, interventional therapy or surgical treatments, these treatments are still symptomatic treatments,

so the effect is less effective. Based on the evidences and logic of human pathology, human anatomy, cell biology, physics, mathematics and chemistry, I have clarified that tunica media dysfunction induced connective tissue remodeling is the main mechanism of atherosclerosis, aneurysm or stiffening<sup>1-34</sup>. This new theory can explain almost all the phenomena of large and medium blood vessels<sup>1-34</sup>. But the traditional vascular hypotheses cannot do it. My previous papers have described the possible etiological treatments of vascular diseases<sup>1,2,8,16</sup>, and this article would not repeat it again. This paper would mainly introduce a better surgery to relieve atherosclerotic disease or hemangioma (Figure 1).

Vascular dysfunction is the main cause of vascular diseases, and vascular tunica media dysfunction (Decrease of the number / function of smooth muscle cells) is the main cause of atherosclerotic disease and aneurysm. However, the traditional vascular treatments mainly focused on the intima of blood vessels, which could not fundamentally solve vascular diseases, so the traditional drug treatments, interventional treatments or surgical treatment were less effective. Based on the theory of tunica media dysfunction, the "**adventitia reinforcement surgery**" proposed in this paper would more effectively treat vascular diseases such as atherosclerotic disease and hemangioma. The main idea of the surgery is as follows.

For severe atherosclerotic disease / hemangioma, the perivascular adipose tissue (vascular "Kuiper Belt") is blunt separated, the diseased blood vessels are tensional wrapped with non-toxic, biocompatible, non-degradable and malleable materials, such as polyethylene. After wrapping the blood vessels, the sealing device is applied to make the materials closely fit the blood vessels and remove the redundant materials, Then the perivascular adipose tissue (vascular "Kuiper Belt") is closed (Figure 1). With the theory of vascular simple harmonic

motion (SHM)<sup>16</sup> and tunica media dysfunction<sup>2</sup>, the connective tissues (atherosclerotic plaques) in the intima would gradually become desertification, be phagocytized and cleared by macrophages, which could relieve the narrowed lumen (Figure 1). **Adventitia reinforcement surgery** applies to arteries, veins and artery / vein grafts.

### References

1. Wang X, Ge J. Myofibroblast Forms Atherosclerotic Plaques. bioRxiv:2020.07.20.212027. DOI: 10.1101/2020.07.20.212027.
2. Wang X, Sun A, Ge J. Medial Injury/Dysfunction Induced Granulation Tissue Repair is the Pathogenesis of Atherosclerosis. arXiv:2010.06683. DOI: 10.48550/arXiv.2010.06683.
3. Wang X, Ge J. Haemodynamics of atherosclerosis: a matter of higher hydrostatic pressure or lower shear stress? Cardiovasc Res 2021;117(4):e57-e59. DOI: 10.1093/cvr/cvab001.
4. Wang X, Ge J. Atherosclerotic Plaque Healing. New England Journal of Medicine 2021;384(3):293-293. DOI: 10.1056/NEJMc2033613.
5. Wang X, Ge J. Spontaneous Coronary-Artery Dissection. New England Journal of Medicine 2021;384(11):1077-1077. DOI: 10.1056/NEJMc2100339.
6. Wang X, Ge J. Hypertension Aggravates Atherosclerosis: A Matter of Pressure Remodeling of Myofibroblasts or LDL Accumulation? J Am Coll Cardiol 2021;77(20):2619-2620. DOI: 10.1016/j.jacc.2021.03.305.
7. Wang X. Macrophages Transform into Foam Cells by Phagocytosing Tissues Formed by Myofibroblasts. Researchgate:354508855. DOI: 10.13140/RG.2.2.26707.91680/2.
8. Wang X. Promising Etiological Treatments of Artery Diseases. Researchgate:354507399. DOI: 10.13140/RG.2.2.15802.72641/1.
9. Wang X. Steroid Hormones Affect Vascular Diseases through Myofibroblasts.

- Researchgate:354534894. DOI: 10.13140/RG.2.2.17257.62565.
10. Wang X. Atherosclerosis. Researchgate:354706286. DOI: 10.13140/RG.2.2.28605.18402/1.
  11. Wang X. In Situ Venous Atherosclerosis. Researchgate:355339577. DOI: 10.13140/RG.2.2.11589.99040.
  12. Wang X. Types of Vascular Media Dysfunction and Possible Risk Factors. Researchgate:355486212. DOI: 10.13140/RG.2.2.16052.99202.
  13. Wang X. Muscle Dysfunction and Myofibroblasts Remodeling in Cardiovascular Diseases. Researchgate:355910950. DOI: 10.13140/RG.2.2.29958.09284.
  14. Wang X. Intimal Foam cells and Perivascular Adipocytes are Essentially Similar. Researchgate:356972652. DOI: 10.13140/RG.2.2.29364.17284.
  15. Wang X. Perivascular Adipocytes are Components of Adventitia: Vascular "Kuiper belt". Researchgate:357025934. DOI: 10.13140/RG.2.2.34338.35520.
  16. Wang X. Physical Principles of Vascular Developing and Remodeling. Researchgate:357220855. DOI: 10.13140/RG.2.2.20693.76003.
  17. Wang X, Tian S. Principle of Transformation between Fibrous Connective Tissue and Adipose Tissue. Researchgate:357657830. DOI: 10.13140/RG.2.2.17756.10883.
  18. Wang X, Tian S. Principles of Blood Vessels to Ligaments Transition. Researchgate:357831361. DOI: 10.13140/RG.2.2.25984.15363.
  19. Wang X. Pathogenesis of Vascular Chronic Total Occlusion (CTO). Researchgate:357877694. DOI: 10.13140/RG.2.2.29831.11687.
  20. Wang X, Tian S. Fundamentals for the Difference of Tissue Structure between Large Elastic Arteries and Muscular Arteries. Researchgate:358021110. DOI: 10.13140/RG.2.2.19594.39367.
  21. Wang X, Tian S. Mechanism of Calcification in Cardiovascular Diseases. Researchgate:358646522. DOI: 10.13140/RG.2.2.13653.70885.
  22. Tian S, Wang X. Physical Remodeling of Connective Tissues. Researchgate:359363655. DOI: 10.13140/RG.2.2.11453.33763.

23. Wang X. Tunica Media Dysfunction and Primary Hypertension. Researchgate:359847538. DOI: 10.13140/RG.2.2.25418.24000.
24. Tian S, Wang X. Hyperlipidemia is the Compensatory Response to the Utilization Rate Decrease of Nutrients: Lipids are Not the Culprit. Researchgate:360069687. DOI: 10.13140/RG.2.2.16433.25444.
25. Wang X, Tian S. Pathogenesis of Atherosclerosis and Xanthoma in Familial Hypercholesterolemia Patients. Researchgate:360318586. DOI: 10.13140/RG.2.2.29335.65445.
26. Wang X, Tian S. Mechanism of Metabolic Syndrome. Researchgate:360484058. DOI: 10.13140/RG.2.2.29997.56801.
27. Wang X. Main Defects in Studies of Lipid-Lowering Drugs: Metabolic Diseases Rather than Lipids is the Risk Factor of Atherosclerotic Diseases. Researchgate:360539761. DOI: 10.13140/RG.2.2.24676.04485.
28. Wang X. Mechanism of Dyspnea in Patients with Pericardial Effusion. Researchgate:360630351. DOI: 10.13140/RG.2.2.20884.78720.
29. Wang X, Tian S. Anticoagulation may be More Effective than Antiplatelet in People at High Risk of Myocardial Infarction or Cerebral Infarction. Researchgate:360713365. DOI: 10.13140/RG.2.2.16913.30562.
30. Wang X. Is Atherosclerotic Plaque Rupture Necessarily Detrimental? -Not Always. Researchgate:360724669. DOI: 10.13140/RG.2.2.18381.31209.
31. Wang X, Tian S. Mechanism of Vascular Occlusion or Stenosis in Atherosclerosis. Researchgate:360823422. DOI: 10.13140/RG.2.2.23027.81445.
32. Wang X. Lipids Metabolism Dysfunction and Vascular Diseases. Researchgate:361510693. DOI: 10.13140/RG.2.2.17887.74400.
33. Wang X. Myocardial Bridge. Researchgate:362413280. DOI: 10.13140/RG.2.2.15781.58083.
34. Wang X. Improvement of Vein Grafts in Coronary Artery Bypass Grafting (CABG). Researchgate:362491790. DOI: 10.13140/RG.2.2.31818.24009.