ADAPTIVE RESPONSES OF ARTERIES

Adaptive responses of arteries and healing of arterial injuries are important in maintaining the structure and function of the arterial tree. These normal responses to altered biomechanical conditions may result in alterations in artery lumen diameter and wall thickness. Abnormal pathologic responses of arteries also include alterations in lumen diameter and wall thickness with lumen stenosis, aneurysm formation, and anastomotic intimal hyperplasia. It is important to understand normal adaptive responses of arteries, because it should help us understand abnormal responses that result in unfavorable conditions leading to clinical complications.

During embryologic development, arteries with high flow grow and enlarge, whereas those with little flow become smaller, atrophy, and disappear. During growth and development, artery lumen diameter is determined by flow whereas wall thickness is determined by wall tension (the product of pressure and radius). This difference is apparent in comparing the pulmonary artery and aorta in the perinatal period. During fetal growth the aorta and pulmonary artery are equal in diameter and thickness. However, at birth, the lungs expand and pressure in the pulmonary artery falls whereas pressure in the aorta rises. Volume flow in the two vessels remains the same, but because of the difference in pressure a marked difference in the thickness of the two vessels develops. In adult life arterial wall thickening occurs in response to increases in pressure. In patients with hypertension, arterial and arteriolar wall thickening develops as an adaptive response to the increase in wall tension. This occurs primarily by intimal thickening. Similarly, when one performs a distal bypass one increases wall tension by increasing pressure, as well as producing a large increase in lumen radius at the anastomotic site. The resulting increase in wall tension would be expected to stimulate intimal thickening as an adaptive response to the increase in tension. The factors that differentiate a normal adaptive intimal thickening from an inappropriate intimal hyperplastic response with lumen stenosis at a vascular anastomosis are unclear and deserve further study.

Arteries exposed to increases in flow such as those feeding an arteriovenous fistula increase in size to accommodate the increase in flow. Similarly, arteries exposed to decreases in flow, such as vessels feeding an amputated limb, will decrease in size. Experimentally produced arteriovenous fistulas demonstrate that proximal artery enlargement occurs to the point at which wall shear stress is normalized. Wall shear stress normally ranges between 10 and 20 dynes/cm² throughout the arterial tree and is the same in large arteries and small capillaries. Wall shear stress appears to be the regulating signal that determines arterial size. Atherosclerotic arteries also are capable of enlarging in response to increases in wall shear stress. A developing intimal plaque produces a focal lumen stenosis resulting in a local increase in wall shear stress. This in turn may stimulate artery dilation to normalize wall shear stress. It has been shown in atherosclerotic human arteries that enlargement can prevent lumen stenosis to the point at which the intimal plaque area occupies approximately 40% of the internal elastic lamina cross-sectional area. Artery enlargement may be unable to prevent lumen stenosis when plaques exceed 40% of the internal elastic lamina area. However, different artery segments respond differently to increasing intimal plaque. In the distal left anterior descending coronary artery, arterial enlargement occurs at a rate faster than the rate of increasing intimal plaque. In the distal left anterior descending coronary artery, arterial enlargement occurs at a rate faster than that of the lumen area in the most severely diseased arteries. Thus it appears that the development of lumen stenosis, the maintenance of a normal lumen cross-sectional area, or the development of an increase in lumen diameter is determined by the relative rates of plaque growth and artery enlargement. Further study of this phenomenon of artery enlargement in response to increasing intimal plaque is needed. Particular attention should be paid to arteries other than the coronary arteries that are prone to atherosclerosis such as the carotid arteries, aorta, and peripheral vessels.

Thus we need a better understanding of normal adaptive responses of arteries, as well as an understanding of the differences between normal adaptive responses and abnormal responses that can progress to pathologic conditions of stenosis and aneurysm. Artery wall responses in atherosclerosis are as important as the process of plaque formation in determining clinical consequences and thus deserve comparable attention and study.

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REFERENCES


