

Surgery of the Aorta and Its Branches

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Within the last 25 years, with improved methods of examination and treatment and better understanding of the functional alterations, it has become possible to diagnose and treat arterial disease before ischemic tissue is irreparably damaged. Two fundamental lesions of the aorta or its main branches are amenable to surgical correction: aneurysm and occlusive disease. We will consider briefly some aspects of the epidemiology and pathophysiology of arterial disease and at greater length the anesthetic and surgical problems associated with the more commonly treated lesions.

Epidemiology of Arterial Disease

Syphilis, once a major cause of arterial disease, has declined in importance, perhaps because of the availability of means of earlier diagnosis and better treatment. However, syphilis still constitutes the basis for formation of about 5 to 10 per cent of aneurysms, especially in the ascending aorta, less commonly in the descending thoracic aorta. Trauma, Marfan's syndrome (a hereditary disease characterized by arachnodactyly deformities of the thoracic cage, dislocated lens, and often, thoracic aneurysms), Takayasu's or "pulseless" disease (a bizarre connective tissue disease characterized by stenosis or occlusion of vessels arising from the aortic arch), and other rarer causes of degenerative change in the walls of blood vessels together constitute another 10 per cent of arterial disease, especially in younger patients. The large residue of arterial disease accounts for more morbidity and mortality in the sixth and seventh decades of life, especially in males, than all other dis-

eases combined.¹ At one time, all degenerative arteriopathies were collectively known as "arteriosclerosis," and even though they have been extensively classified according to their pathologic features,² the term is still used loosely to apply to all arterial disease. In the following discussion "atherosclerosis" refers to lesions of large and medium-sized arteries, characterized by intimal deposits of yellowish plaques rich in lipids and cholesterol. "Arteriosclerosis" refers to more diffuse sclerotic lesions of smaller arteries and arterioles, affecting any arterial coat, but especially the media. Although still thought to be an inevitable accompaniment of aging (fig. 1), the causes of arteriosclerosis and atherosclerosis are not established, and there is much debate regarding the importance of certain etiologic factors.

Genetic factors are strong determinants of at least three factors predisposing to atherosclerosis; serum lipids, blood pressure, and blood sugar.⁴ For the last 50 years a relationship between diet, blood cholesterol level, and formation of atheromatous plaques, so significant that it was at one time suggested that all fats be avoided, has been evident. More recently, it has been shown that a diet rich in saturated fatty acids will elevate serum cholesterol and phospholipid levels in all subjects, whereas unsaturated fatty acids will decrease these levels.⁵ Most significantly, in rare cases of precocious atherosclerosis affecting children, plasma lipids are markedly elevated.⁶ No direct relationship between the consumption of moderate amounts of alcohol and arterial disease has been shown.

Cigarette smoking is said to accelerate the rates of dying from all causes in some unknown manner. Buerger's disease (thromboangiitis obliterans) is still believed directly related to cigarette smoking,⁷ but its incidence has declined to the point where its very existence has been questioned.⁸ Although cigarette smoking is said to be strongly associated

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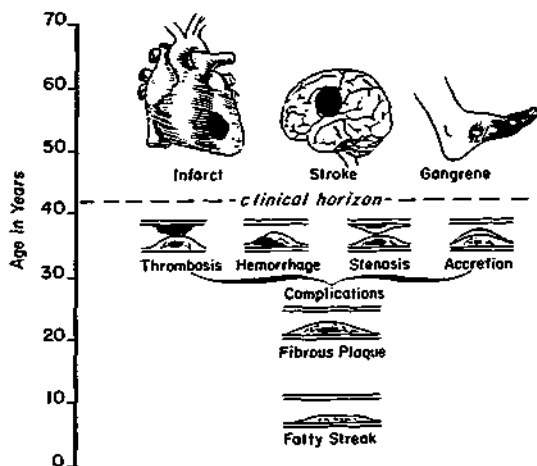


FIG. 1. Stepwise development of atherosclerosis and some of its effects. Note that the earliest change, fatty streak to fibrous plaque, occurs many years before a typical atheroma becomes established, and even longer before it becomes symptomatic. (Courtesy of Holman *et al.*²)

with coronary artery disease, Wilens and Flair found no statistical difference between the incidences of myocardial infarction at necropsy in heavy smokers (25.6 per cent) and non-smokers (20.5 per cent).⁹ On the other hand, numerous reports based on clinical studies and mortality statistics supporting a relationship between smoking and coronary disease are summarized in a recent Public Health Service Report.¹⁴ The discrepancy between clinical data and necropsy reports may lie in the large percentage of myocardial infarcts not evident during life; for example, 51 per cent of acute and 80 per cent of healed infarcts at Bellevue Hospital,¹¹ and 4 per cent of acute and 50 per cent of healed infarcts at the Mayo Clinic.¹² If "silent" infarcts were common in non-smokers, the apparent discrepancy would be resolved.

About 20 per cent of all patients with obliterating atherosclerosis of the lower extremity have *diabetes mellitus*, and of all the complications of diabetes, arteriosclerosis is the most common. Obviously, it occurs more frequently and is more severe in the elderly patient, but it is not uncommon in uncontrolled juvenile diabetics.¹³

About 10 per cent of the world population have, or will develop, *hypertension*, either

essential or secondary to other diseases, especially renovascular disease. Evidence suggests that hypertension increases the rate of development of atherosclerosis, as well as the incidences of cerebrovascular accidents and myocardial infarction.¹⁴

Anesthetic Considerations in Arterial Surgery

"The manifestations of atherosclerosis should not be considered as evidence of heart disease, aortic disease, renal disease or brain disease. It is all part of the same process, which frequently proceeds at different rates in different parts of the arterial tree. This approach is essential to the modern conception of the prevention and treatment of the total disease process."¹⁵ This simple statement eloquently states the most important principle in anesthetic management. Knowledge that arterial disease is a systemic disease and that only its localized manifestations bring patients to the operating room should alert the anesthesiologist to look for manifestations in other organ systems. The protean nature of this disease is well illustrated in table 1, which shows the incidences of related diseases in patients operated upon in our institutions for localized lesions.