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Robert C. Horn Jr.

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EDITORIAL

Coronary Artery Disease and Myocardial Infarction

Robert C. Horn, Jr.

The failure of the coronary arteries to supply the myocardium with an "adequate" supply of oxygenated blood is the direct cause of a vast amount of human disease, all too frequently fatal. Almost without exception the inadequate coronary arteries are the site of advanced arteriosclerosis or atherosclerosis. The coronary arteries differ from most other arteries of the human body, both functionally and anatomically; in particular, they have a distinct intimal layer, which contains longitudinal smooth muscle fibers and which thickens progressively throughout life, beginning almost from the time of birth. Arteriosclerosis occurs earlier and more extensively than in most other vessels of the body, differing from the process elsewhere only in the great frequency of calcification and in the almost invariable vascularization of the sclerotic intima. Probably most of these intimal vessels arise from the vasa vasorum, although some undoubtedly penetrate from the arterial lumen. Coronary artery sclerosis and stenosis is of greatest extent and severity in the the first 2 to 3 cm. of each of the three major extramural trunks. In the great majority of hearts bearing myocardial infarcts, at least two major trunks show reduction of the cross-sectional area of their lumina by an estimated two thirds to three fourths or more.

By careful routine pathologic study (not including serial sections) thrombi can be demonstrated to complicate the coronary arteriosclerosis in almost half of infarct-bearing hearts, hemorrhage in atheromatous plaques somewhat less often.[1] More than one vessel may be involved by either process, and the two processes may occur in the same heart or even the same artery, at times in anatomic continuity with one another. Both thromboses and hemorrhages occur in vessels with lumina already greatly compromised by atherosclerosis. Most arterial intramural hemorrhages are small, suggesting that they belong among the degenerative phenomena of arteriosclerosis. However,

From the Department of Laboratories, Henry Ford Hospital, Detroit, Michigan.
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the extent of some hemorrhages is consistent with the belief that they may make a significant contribution to arterial narrowing, and the anatomic relationship of some hemorrhages and thrombi supports the belief that there may be a causal relationship. Although arterial intramural hemorrhage unquestionably may at times precipitate thrombosis, it seems doubtful that this mechanism accounts for a large proportion of coronary artery "occlusions." It has been suggested[2] that even small intramural hemorrhages may be significant in triggering arterial spasm. In roughly one fourth to one third of myocardial infarcts, the only coronary arterial lesions are old ones of atherosclerosis; thrombosis and hemorrhage, when they do occur, are so regularly associated with such an advanced degree of arteriosclerotic narrowing that the observer frequently cannot help wondering what difference the minimal added insult might have made.

Thrombi are more often seen in association with recent myocardial infarcts, whereas in the case of healed infarcts arteriosclerosis is more extensive and severe and is less frequently complicated by thrombosis or hemorrhage. Likewise, fresh infarcts are seen at autopsy more frequently in women and in younger men, healed infarcts in older men. These facts suggest that some of the changes classified as arteriosclerotic in association with healed infarcts are really the result of organization of trombi or, perhaps, of intramural hemorrhages. With the passage of time it becomes increasingly difficult to distinguish histologically between organized thrombi and organized hemorrhages and also atherosclerotic plaques.

To return to the opening sentence of this editorial — what is an "adequate" supply of blood to the myocardium? — although lesions producing "significant narrowing of the coronary arterial lumina can be found in the vast majority of instances of myocardial infarction, cases occur in which little narrowing is apparent. Sometimes, but not always, alternative explanations may be available, such as the demand for an increased blood supply of a hypertrophied heart of hypertensive or rheumatic disease. Many infarcts are associated with total, or complete, occlusion of the artery supplying that portion of the myocardium. Many more, however, show a degree of occlusion varying from nearly total to roughly two thirds. Total occlusions with thrombosis are seen more often at autopsy in association with recent infarcts; yet 85 per cent of patients with fresh myocardial infarction survive.[2] On the other hand, sudden, unexpected "coronary" death is most often associated with an old, healed myocardial infarction without total coronary artery occlusion or any demonstrable acute event. Conversely, the pathologist sees, not rarely in the autopsy room, undamaged hearts with arteries as sclerotic and narrow as many of those productive of myocardial infarcts, at times even with complicating thromboses or hemorrhages.

In many instances the cause-and-effect sequence of events is clear: coronary arteriosclerosis with stenosis, → thrombosis, → total occlusion, → myocardial infarction. So many cases of fatal coronary artery disease do not present this clearcut picture, however, that one must seek an additional factor or factors not apparent on pathologic study — collateral circulation, alterations in cardiac output, spasm, arrhythmia, or other — to explain why, with apparently comparable limitations of coronary blood flow, one patient will and another will not suffer acute myocardial infarction and why, of two people dying suddenly with coronary artery disease, autopsy will disclose an acute event in one but only evidence of healed, or old, disease in the other.

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