

SOME FACTORS IN THE CAUSATION OF INTIMAL HÆMORRHAGES AND IN THE PRECIPITATION OF CORONARY THROMBI*

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THE liberation of thromboplastic substances from lesions in the walls of arteries, particularly those affected by advanced atherosclerosis, may be taken as the immediate cause of precipitation of arterial thrombi. Other factors take part in the process, eddying and stagnation of blood at points of atherosclerotic stenosis, stasis of blood due to shock and other causes, and increased viscosity and coagulability of the blood, but without the primary injury to the arterial wall it is doubtful if thrombus formation can occur. This paper is concerned solely with the mechanism of production of intimal hæmorrhage, an injury which is admitted now to be the common precipitating cause of coronary thrombosis.

Intimal hæmorrhages have been noted repeatedly in the past at the site of precipitation of coronary thrombi, but they were considered to be the result either of rupture of the vasa vasorum due to inflammation¹ or of back flow of blood from the lumen into the intima through a defect produced by the rupture of an atheromatous "abscess".² The associated thrombosis in the arterial lumen was regarded, therefore, as being due either to an inflammatory process or to the contact of blood with the raw surface of an atheromatous ulcer, and not to the intimal hæmorrhage *per se*.

In a report in 1936^{3a} and in subsequent reports,^{3b,c,d,e} I showed by serial sections that intimal hæmorrhages in coronary arteries are intrinsic lesions; inflammatory changes were absent in most cases, and often there was no break in the tissues lying between the hæmatoma and the lumen of the artery. The hæmorrhages were found to be due to the rupture of capillaries which are derived from the coronary lumen. This finding has been confirmed and elaborated upon by Wartman,⁴ Winternitz and

his co-workers,⁵ and Horn and Finkelstein.⁶ Intimal capillaries are not normal structures; they are found only in sclerotic arteries, and they develop, apparently, in response to the demand for nutrition by the plaques of atherosclerosis in which they ramify (Fig. 1). They are not related to the usual vasa vasorum although they do anastomose with these structures in some cases.

The rupture of intimal capillaries and the subsequent formation of intimal hæmorrhages (Fig. 2) is a fairly common finding in sclerotic coronary arteries; and it may lead to a variety of sequela, some of which are disastrous. These secondary phenomena have been discussed in detail elsewhere^{3b,c} and they may be summarized as follows. (1) Occasionally a small intimal hæmorrhage which is otherwise innocuous may irritate the medial coat and result in spasm and acute coronary insufficiency. (2) In a few cases (13 per cent in my series) the hæmorrhage attains such a size that the coronary lumen is obstructed by pressure, and sudden death from coronary insufficiency results. (3) In approximately one-third of the cases the only result is a slight increase in the size of the atherosclerotic plaque, first from the addition of blood to its bulk, and later from the presence of products of repair. (4) Finally, in about half the cases secondary thrombosis occurs in the coronary lumen^{3b,c,d} (Fig. 3).

The liberation of thromboplastic substances from an intimal hæmorrhage may occur in a variety of ways. When the hæmorrhage is superficial there may be diffusion of blood and thromboplastic substances from the intima into the lumen. The rupture of the nutrient capillaries may be so extensive that necrosis of the intima results. The necrosis may actually involve the endothelium, or the pressure of blood within the lumen may rupture the thin shell of viable tissue, thus producing a defect with a raw surface. When the hæmorrhage occurs into the deeper intimal layers the capillaries adjacent to the point of rupture may thrombose; retrograde thrombosis may then take place, and,

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