

timal hemorrhages (26 per cent). When the comparison is confined to cases of marked coronary sclerosis alone the hypertensive group still shows a definite preponderance of intimal hemorrhages over the non-hypertensive group (82 per cent versus 59 per cent). Elimination of the cases of indefinite hypertension from the calculation has no appreciable effect.

When blood pressure readings were available in the hypertensive groups, as they were in the majority, there was a consistently higher reading in the group with intimal hemorrhages (an average of 183 mm. systolic and 103 diastolic in 28 cases) than in the group without intimal hemorrhages (an average of 169 mm. systolic and 90 mm. diastolic in 18 cases). As can be seen from the calculated standard deviations the blood pressure readings were widely scattered in both groups. In a small series like this they can be taken as indicating a trend rather than as being statistically significant.

The findings in this series suggest strongly that persistent hypertension is intimately concerned in the rupture of intimal capillaries and in the formation of intimal hemorrhages. Horn and Finkelstein⁹ have claimed that if hypertension is a common cause of coronary occlusion there would be a greater incidence of coronary occlusion in hypertensive individuals. My series includes 70 cases of persistent hypertension; 60 per cent of these had intimal hemorrhages, the common cause of coronary occlusion, and 33 per cent had occlusive phenomena. In this short series, then, the high incidence of intimal hemorrhage and coronary occlusion in hypertensive persons is clearly demonstrated. It shows, further, that the higher the blood pressure level the greater is the incidence of intimal hemorrhage and occlusion.

This study has been concerned solely with the influence of persistent hypertension on coronary occlusion. But one can safely assume that, if persistent high blood pressure can disrupt intimal capillaries, the temporary rises in blood pressure caused by violent emotion or exercise will have a similar effect.

Interesting examples of the possible effects of transient hypertension were encountered in my series. Two elderly men were admitted to hospital with fractured skulls and with evidence of progressively increasing intracranial pressure. Their blood pressures before their accidents were said to be normal and their

hearts at autopsy were small; but their blood pressures rose to a systolic level of 240 mm. and 170 mm., respectively, shortly before death and remained at these levels for several hours. At autopsy each case showed a moderate grade of coronary sclerosis with small recent intimal hemorrhages into atherosclerotic plaques. Five additional cases have been encountered in which there was a temporary elevation of blood pressure prior to death due to intracranial injury. None of these showed intimal hemorrhages, but it is noteworthy that on the whole they showed a lesser grade of coronary sclerosis and their blood pressures did not rise to so high a level as in the two cases given above. It cannot be stated definitely that the intimal hemorrhages in the two positive cases were due to temporary hypertension from intracranial injury, but the evidence is suggestive. I believe that transient hypertension caused by excessive exertion or violent emotion will have a similar effect, and that these activities should be avoided by all patients with coronary artery disease. This belief agrees with present-day clinical opinion.

CAPILLARY FRAGILITY

Variation in the strength and elasticity of capillary walls also plays a part in the integrity of intimal capillaries and in the etiology of intimal hemorrhages. It would appear that capillary fragility may be increased in several ways.

Local inflammatory changes in the arterial wall, or toxic influences of a more general nature, may affect the normal ability of endothelial cells to contract and dilate. Histologically, inflammatory infiltration in regions of intimal hemorrhage is not infrequent, but it is absent in many cases and, when present, appears to be related to the disintegration of red cells and to the necrosis of tissue, and is probably secondary.

The influence of age on the elasticity of capillary walls may also be important in the causation of intimal hemorrhage. Cutter and Marquardt¹² have noted an increase in fragility of capillary walls in direct proportion to the age of the patient.

Increased capillary fragility from defective nutrition is another possible etiological agent, and in this regard one thinks immediately of avitaminosis C. The mode of action of vitamin