calcific deposits should help to strengthen the supporting stroma and prevent capillary dilatation and rupture, but it is noteworthy that calcification in the cerebral arteries is rare, and none at all was seen in this series.

Finally, the strength and elasticity of the walls of capillaries in cerebral arteries may be affected by a number of factors. It is known that capillary fragility increases with advancing age, vitamin deficiency or local inflammatory conditions. Each of these factors may be of importance in the production of intimal hemorrhage in certain cases.

SUMMARY

Intimal hemorrhages in sclerotic cerebral arteries are similar in structure to those described previously in sclerotic coronary arteries. They result, not from backflow of blood through defects produced by rupture of atheromatous "abscesses," as was previously thought, but from rupture of capillaries derived from the main arterial lumens.

From the observations in this series it appears that capillary rupture with intimal hemorrhage is intimately concerned with the mechanism of cerebral arterial thrombosis and possibly, in certain cases, with the causation of cerebral arteriospasm and rupture. It is suggested that the factors responsible for the rupture of intimal capillaries in the cerebral arteries are high intracapillary pressure from hypertension, progressive atheromatous degeneration of the supporting tissues and increased capillary fragility from a variety of causes.