Ellis [10] found that the miliary aneurysms which are usually regarded as the cause of cerebral hemorrhage are not true aneurysms but are encapsulated hematomas. This important question is now being studied and will be discussed later.

From the observations in this short series of cases it appears that capillary rupture with intimal hemorrhage is intimately concerned with the mechanism of cerebral arterial thrombosis and possibly with the causation of cerebral arteriospasm and hemorrhage in certain cases. If this is true, the factors responsible for the rupture of intimal capillaries should be the immediate causes of these lesions. The factors responsible for the rupture of intimal capillaries in the coronary arteries have been described elsewhere,[1] and the same factors hold good for the cerebral vessels. The integrity of a capillary wall in any part of the body depends on the pressure of blood within its lumen, the strength and elasticity of its wall, and the rigidity of its supporting stroma. Because intimal capillaries arise directly from the lumen of a large artery, it is argued that they will be subjected to a greater blood pressure than those which lie at the end of a long series of arteries and arterioles. This argument is not new: Ellis [10] stated that "the small vessels of the basal ganglia, notoriously the site of hemorrhage, are direct branches of the cerebral arteries and hence are subjected to a blood pressure greater than vessels of similar size in other parts of the brain and possibly of the body." It is reasonable to assume that the pressure in intimal capillaries of the cerebral arteries, normally high, will be increased in cases of persistent hypertension or of temporary elevation of blood pressure from excessive exercise or emotion. In these circumstances the strain on the capillary walls will be increased, and there will be imminent danger of capillary dilatation and rupture. In the series reported here, persistent hypertension was present in 3 of the 4 cases in which intimal hemorrhage was observed at the site of precipitation of a thrombus. Aring and Merritt [9] investigated 96 cases of cerebral thrombosis and found that in 85 per cent the systolic pressure was in excess of 140 mm. of mercury and that in 50 per cent the diastolic pressure was in excess of 100 mm. of mercury.

Loss of rigidity of the tissues supporting the capillary walls appears to be equally important in the causation of capillary rupture in cerebral arteries. Each of the intimal hemorrhages in this series was found in an area of atheromatous degeneration; the softening process which is characteristic of atheroma allows the capillaries to dilate, and if the intracapillary pressure is high enough, overdilatation and rupture may occur. Theoretically, replacement of the atheromatous material by

10. Ellis, A. G.: Publ. Jefferson M. Coll. & Hosp. 8:1, 1915.