hemorrhages. Of particular interest is their relation to arteriospastic attacks in hypertensive persons as suggested in case 1 in this series. The clinical signs of cerebral arteriospastic attacks over a period of four years in this case and the postmortem observation of 12 distinct intimal hemorrhages of varying ages in the larger cerebral arteries suggest that they were related. Many of the hemorrhages lay at the outer borders of atheromatous plaques in proximity to the media (fig. 1 *A*), and it is reasonable to suppose that the sudden disruption of tissue by the hemorrhages may have set up transient spasms of the muscle coat. The observations of others tend to confirm this hypothesis. The walls of cerebral arteries are known to be supplied by vasomotor fibers, and local spasm due to local injuries or influences definitely occurs. Stroking the adventitia of a pial artery with a blunt instrument at operation causes spasm of that part of the vessel.[6] Also, injuries to arterial walls are known to cause pain. Waterston[7] found that the contact of the point of a needle with the wall of an artery elicited sharp pain, and when the needle point was pushed into the wall a peculiar sickening pain, associated with nausea and faintness, resulted. Feiling,[8] and Aring and Merritt[9] commented on the frequency with which certain prodromal symptoms occur in patients with cerebral arterial thrombosis. The symptoms consist of headache, dizziness, transient weakness of one or both limbs or of one side of the face, temporary aphasia and other symptoms. Sometimes slight hemiplegic weakness may appear and rapidly vanish, to be followed a few days later by severe hemiplegia. It is possible that these premonitory symptoms in certain cases, including pain in the head, are due to arteriospasm from irritation by the intimal hemorrhages which precede and apparently cause cerebral thrombosis. It must be admitted, however, that attempts to demonstrate nerve fibers in the actual area of intimal hemorrhage have as yet been unsuccessful.

Equally hypothetic is the relation of intimal hemorrhage to cerebral arterial rupture. An accidental finding in a case in my series was a large intimal hematoma of a middle cerebral artery which had broken through the thin medial layer and lay between the adventitial fibers (fig. 3). It is possible that a true intracranial hemorrhage would have occurred in this case if the process had continued. Cases of classic cerebral hemorrhage due to rupture of the lenticulostriate artery have not as yet been studied, but the observations of others suggest that in this vessel also the precursor of rupture may be an intimal hematoma.

6. Penfield, W.: Personal communication to the author.
7. Waterston, D.: Lancet **1**:943, 1933.
8. Feiling, A.: Practitioner **133**:62, 1934.
9. Aring, C. D., and Merritt, H. H.: Arch. Int. Med. **56**:435, 1935.