

Primary Intraventricular Hemorrhage in Adults

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SUMMARY Five adults with primary intraventricular hemorrhage are described. The presenting features included headache, confusion and drowsiness. Focal neurological signs were minimal or absent. All five had a history of hypertension, three patients had bilateral internal carotid occlusion at its origin, one had unilateral occlusion of the left internal carotid artery with severe stenosis of the contralateral siphon. Unilateral occlusion of the middle cerebral artery was present in the fifth patient. Pathological examination of the brain from one patient showed the presence of several hemorrhagic "lacunar" infarcts adjacent to the left lateral ventricle, one showing direct continuity of blood in the lacune with the massive intraventricular hematoma. We hypothesize that such a finding illustrates one possible mechanism for this unusual type of hemorrhage. Patients with longstanding hypertension and severe occlusive disease of the internal carotid arteries may be predisposed to this unusual complication.

Stroke Vol 17, No 5, 1986

THE TERM PRIMARY INTRAVENTRICULAR HEMORRHAGE (IVH) was introduced by Sanders who described hemorrhage into the ventricular system of the brain, without associated "laceration of the ventricular parieties." We describe the clinical features and angiographic findings of five adults with primary IVH and discuss the potential pathogenic mechanism(s).

Patients

Patient One

Mr. J.W. aged 53 presented in October 1983 with an exacerbation of congestive heart failure. On admission, his blood pressure was 160/90, he was disoriented to place and time, agitated and incontinent of urine. Short term memory was impaired. The neurological examination was otherwise normal. He had been taking acetylsalicylic acid 325 mg. q.i.d. Because of the confusion a computerized tomographic (CT) scan (October 14) was performed which revealed hemorrhage within the left lateral ventricle but no parenchymal component to the hematoma.

There was a history of heavy alcohol and tobacco consumption and a past medical history of diabetes, chronic obstructive airway disease, focal motor epilepsy and well-controlled hypertension, the latter for approximately 30 years. In 1980, he had experienced numerous episodes of right amaurosis fugax and one episode of right central retinal artery occlusion. Four vessel angiography at the time demonstrated bilateral occlusion of the internal carotid arteries at their origins. In August 1980, a right superficial temporal to

middle cerebral artery anastomosis was performed. In September 1980, a right hemisphere infarct had resulted in dysarthria and mild weakness of the left face, arm and leg.

The cardiac failure improved with appropriate therapy and the disorientation, agitation, incontinence and memory disturbance resolved. He was readmitted in November 1983 with pneumonia and worsening cardiac failure which was refractory to therapy and he died one week after admission. On the final admission, platelet count was 439,000, prothrombin time was 14.1 sec. (control 11.6) and partial thromboplastin time (PTT) was 28.9 sec.

At autopsy, there was a congestive cardiomyopathy with pulmonary and peripheral edema, bronchopneumonia, and multiple recent and old pulmonary emboli. Hepatic cirrhosis was not identified. There was moderately severe arteriolonephrosclerosis. An old right frontal cortical and white matter infarct and an organizing hematoma within the left lateral and third ventricles were found at brain cutting after appropriate fixation (fig. 1).

The right frontal cystic infarct was in the watershed region between the anterior and middle cerebral artery territories of supply, and on histological sections showed features typical of an old lesion, with the presence of histiocytes containing lipid and old blood pigment, and intense reactive astrocytic gliosis in the surrounding preserved neuroglial parenchyma. Subserial sections of cerebral tissue immediately adjacent to the residual clot in the left ventricle were examined (see upper arrow in fig. 1). The intraventricular blood was in continuity with old blood pigment contained in macrophages within the parenchyma of the fornix. In a section from the wall of the left ventricle, altered blood pigment was identified in the ventricular cavity and several small old hemorrhagic subependymal lacunar infarcts surrounded by patchy gliosis were seen (fig. 2). At the edge of the left lateral ventricle, the intraventricular hematoma was clearly seen to be in continuity with a small hemorrhagic infarct just deep to the ependyma (fig. 3). Other sections from both the right and left basal ganglia and the ventricular cavities showed focal patchy loss of ependyma, hemosiderin-laden macrophages at the surface of the disrupted ependyma,

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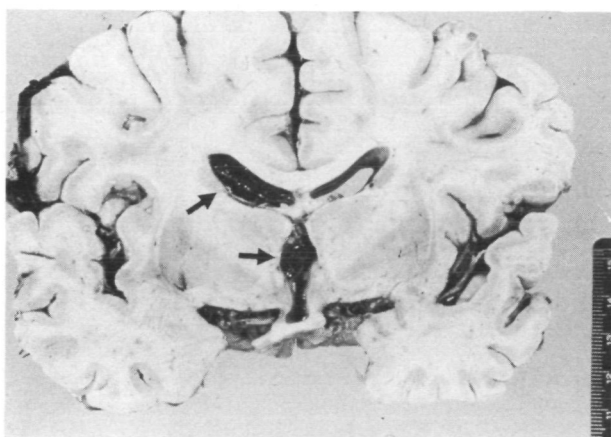


FIGURE 1. Coronal section of cerebral hemispheres at the level of the optic chiasm shows clotted blood in the left lateral and third ventricles (arrows). Communication between ventricular blood and a parenchymal lesion is not readily apparent.

and intense subependymal gliosis. No lacunar infarcts were identified in the thalami or pons.

Other structures examined, including brainstem, cerebellum and spinal cord were normal apart from small numbers of swollen protoplasmic astrocytes, which were seen throughout the grey matter of the brain. Sections of choroid plexus from both lateral ventricles were grossly and microscopically normal. A few small vessels in the cerebral hemispheres showed hyaline arteriosclerosis and arteriosclerosis, but no Charcot-Bouchard aneurysms were identified and lipohyalinosis was not seen. The EC/IC anastomotic vessel was patent.

Patient Two

Mr. J.D. aged 59 was found unconscious on November 12, 1983. The duration of coma was uncertain. On admission to hospital he was afebrile without neck stiffness. He was semicomatose responding to painful stimuli with verbalization of discomfort. There was a left facial asymmetry and a paucity of spontaneous

movement on the left compared to the right, although the left side moved appropriately to painful stimuli, reflexes were symmetrical and the plantar responses were flexor bilaterally. He had not been on anticoagulants or antiplatelet therapy. A CT scan revealed a large intraventricular hemorrhage. The platelet count was decreased, but the actual number was not stated. The prothrombin time was 10.9 seconds with a control of 11.6. There was a history of mild hypertension and heavy alcohol and tobacco consumption. In 1981 he had experienced three episodes of left monocular amaurosis fugax. Bilateral occlusion of the internal carotid artery origins was demonstrated on four vessel angiography at that time.

The mild left sided weakness that was present on admission resolved within 24 hours, but on discharge seven days later, he remained confused and disoriented with poor short term memory. The confusion subsequently cleared but his short term memory remained mildly impaired on further follow-up six months later.

Patient Three

T.G., a 62 year old man was admitted in March 1982 following the abrupt onset of headache, drowsiness and neck stiffness. His temperature was 38.1, pulse 88, blood pressure 130/74. There was an old left hemiparesis but no new neurological signs other than neck stiffness. The CSF was xanthochromic and the CT scan revealed an intraventricular hemorrhage and mild hydrocephalus. He was neither on antiplatelet nor anticoagulant medications. His platelet count and prothrombin time were normal.

There was a past history of well controlled hypertension, ischemic heart disease, peripheral vascular disease and generalized epilepsy. In 1978, he had suffered a right hemisphere infarct with resultant mild dysarthria and weakness of the left face, arm and leg. Four vessel angiography at the time demonstrated bilateral occlusion of the internal carotid arteries at their origins. He therefore had undergone a right superficial temporal to middle cerebral artery anastomosis.

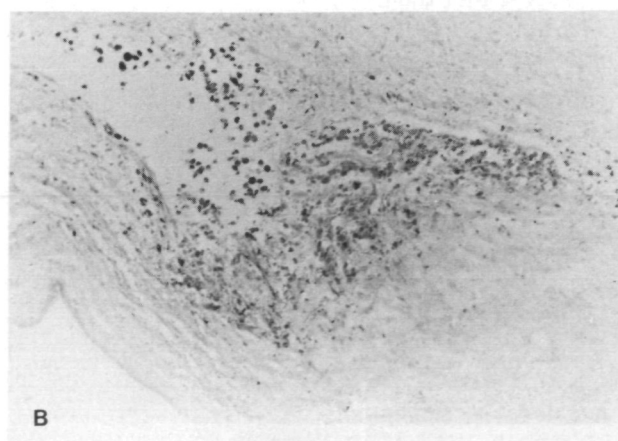
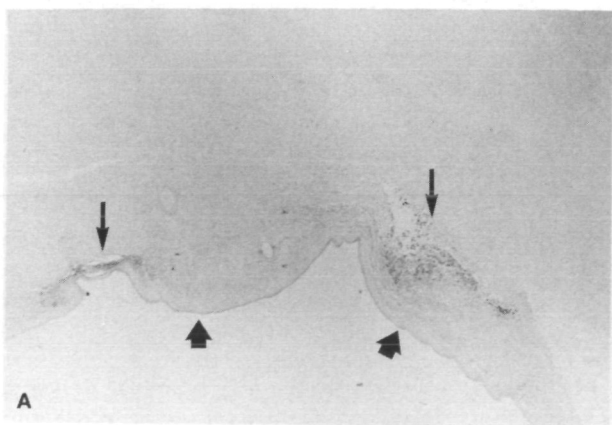


FIGURE 2. (a) Section from edge of the left lateral ventricle shows two small discrete areas of old hemorrhagic necrosis (arrows) immediately adjacent to ependymal lining (arrowheads). Clot has fallen away during processing of tissue. (b) Magnified view of the larger lesion shows many hemosiderin-laden macrophages. (per's Prussian blue stain; A*34; B*140).

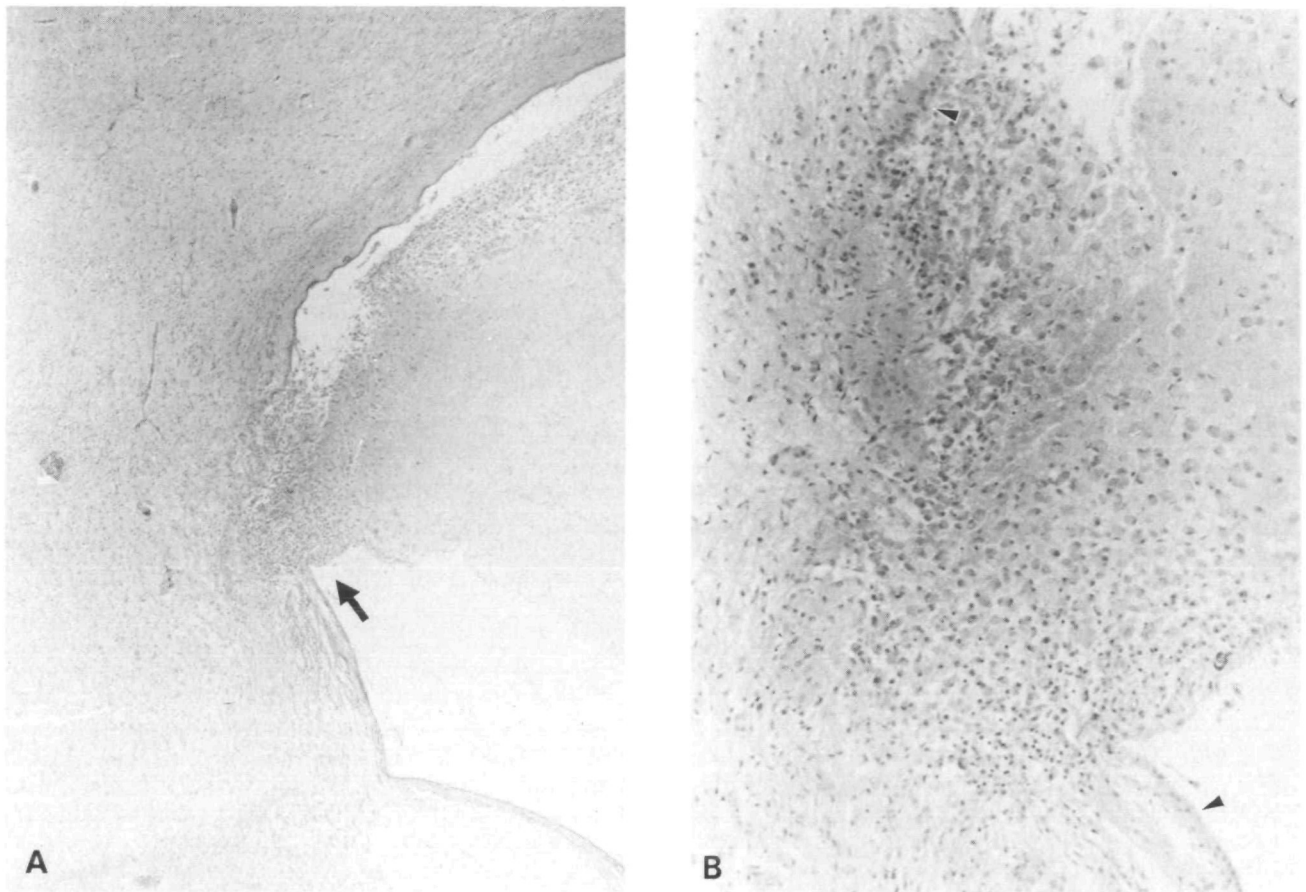


FIGURE 3. (a) Section from wall of left lateral ventricle shows continuity between ventricular clot and subependymal area of hemorrhagic necrosis (arrow). (b) Magnified view of region shown by arrow in (a) shows granular altered blood pigment and hemosiderin-laden macrophages. Ependyma (arrowheads) has been focally disrupted. (hematoxylin and eosin; A*33; B*135).

His headache gradually cleared, but he remained confused and disoriented one week later. At review three months after discharge he had returned to his premorbid state.

Patient Four

R.E. was a 57 year old man who experienced four TIAs over a three month period. These consisted of the sudden onset of pins and needles in the left hand which rapidly spread to involve the whole arm and the left leg without involvement of the trunk. These would last approximately 30–40 seconds and were associated with heaviness of the involved limbs together with slurring of his speech. He was placed on acetylsalicylic acid 300 mg/day. Three weeks after this he suddenly developed severe generalized headache, nausea, vomiting and slurring of his speech similar to the previous episodes. There was no sensory or motor disturbance and the dysarthria rapidly resolved.

On examination he was confused and disoriented without neck stiffness. His pulse was 80, blood pressure 150/80. The neurological examination was otherwise normal. His CT scan revealed intraventricular hemorrhage (fig. 4) and a four vessel angiogram demonstrated occlusion of the left internal carotid at its origin and severe stenosis of the siphon on the right.

The vertebral arteries appeared normal. His platelet count was 408,000. His PT was 100% and the PTT was 26 seconds with a control of 29 seconds.

Patient Five

B.L. was a 53 year old woman who was admitted in November 1978 following the sudden onset one week previously of headache, nausea, vomiting and increasing drowsiness over the ensuing week. On examination, the blood pressure was 150/80 and she was drowsy with neck stiffness. There were no focal neurological signs. Lumbar puncture revealed xanthochromic CSF and a CT scan demonstrated a hemorrhage confined to the left lateral ventricle. Platelet count and clotting studies were normal. Four vessel angiography demonstrated mild stenosis at the origin of both internal carotid arteries and the left supraclinoid internal carotid artery. The left middle cerebral artery was occluded distal to the origin of the anterior choroidal artery.

There was a past history of well controlled hypertension and diabetes. She was a non-smoker, non-drinker and was not on antiplatelet or anticoagulant therapy at the time. Following angiography, she developed weakness of the right arm and leg which improved but never resolved completely. The headache and drowsi-

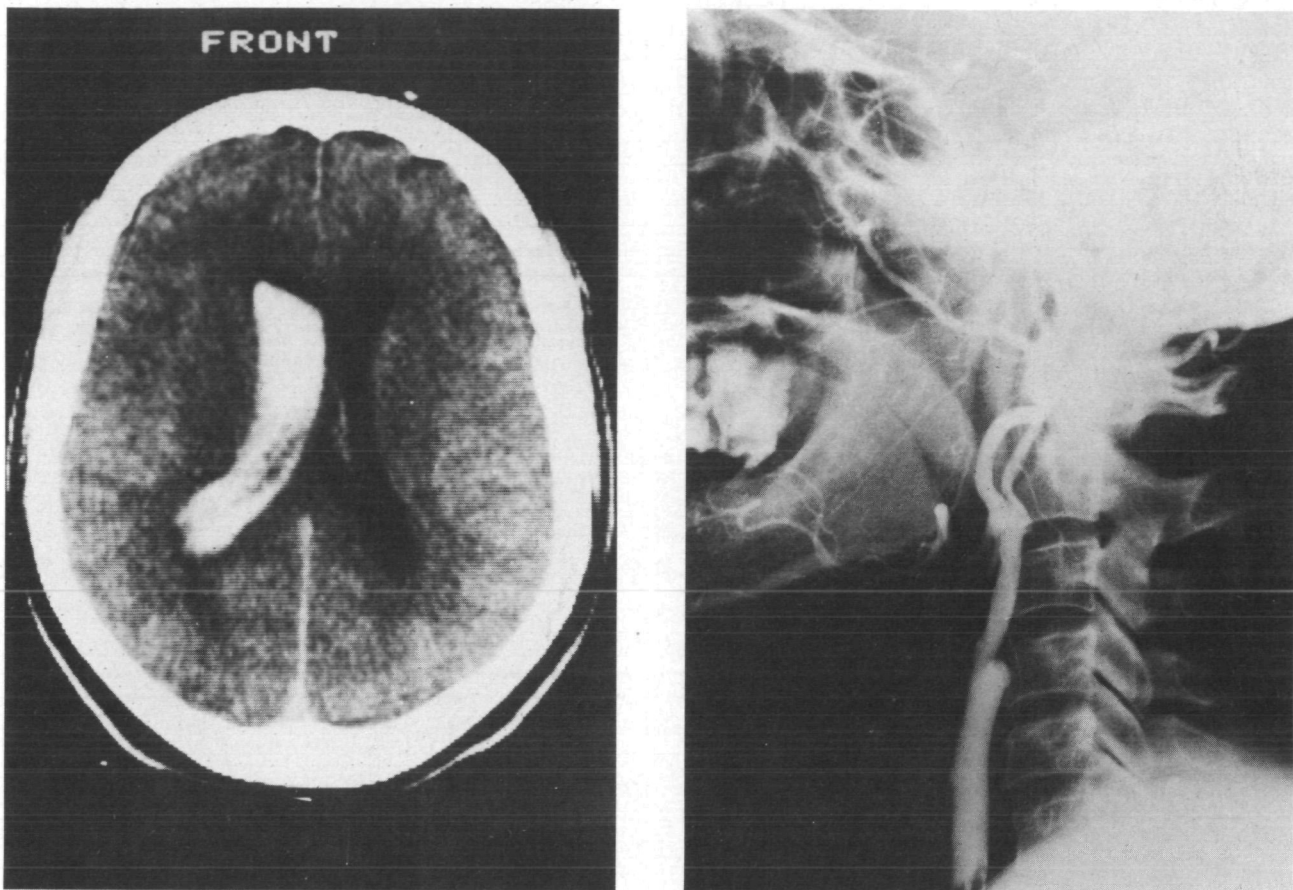


FIGURE 4. (a) CT scan showing hemorrhage within the left lateral ventricle. (b) Angiogram: left internal carotid occlusion and severe stenosis of the right internal carotid siphon.

ness resolved and there were no permanent sequelae that could be ascribed to the intraventricular hemorrhage.

Discussion

Primary intraventricular hemorrhage is uncommon. It has been described with aneurysms, vascular malformations and tumors within the choroid plexus or on the anterior choroidal or lenticulostriate vessels.²⁻¹⁰ It has been reported in a patient with leukemia and attributed to a disorder of coagulation¹¹ and also in a patient with a chromophobe adenoma complicated by pituitary apoplexy.¹² In a number of instances no etiology could be established.¹³⁻¹⁵

The mode of presentation in our patients is similar to that previously described, with the sudden onset of headache, nausea and vomiting together with an alteration of the mental state and/or level of consciousness. Focal neurological signs are either minimal or absent. Focal and/or generalized epilepsy may occur and xanthochromic CSF is the rule. A localized intraventricular hematoma simulating tumor may occur and is thought to form as a result of bleeding into the ventricle under low pressure and subsequent clotting.^{15, 16}

The mechanism of hemorrhage is uncertain. In two patients (2 and 3) an aneurysm cannot totally be excluded as there was a period of two and four years

between angiography and the intraventricular hemorrhage. Aneurysms are known to develop in the presence of carotid occlusion, particularly in patients with Moya Moya disease and bilateral carotid artery occlusion.¹⁷⁻¹⁹ It has been suggested that this is due to increased flow in the non-occluded vessels or "hydraulic imbalance" within the circle of Willis.^{17, 20, 21} Altered hemodynamics have been shown to result in the formation of aneurysms in experimental animals.²² It is interesting to speculate that in the patients with hypertension and severe bilateral occlusive disease of the carotid arteries such altered hemodynamics may be responsible for the intraventricular hemorrhage.

The pathological findings in the first patient described above would suggest, however, an alternate mechanism than rupture of a berry aneurysm. There are subependymal arteries that are branches of the choroidal arteries or rami striate laterales of the middle cerebral artery. Centrifugally directed arterioles arise from these subependymal vessels.^{23, 24} Pathology within these vessels has been implicated in the pathogenesis of periventricular infarcts in neonates. Although Sanders¹ stated that he could not directly trace the IVH to rupture of the arteries ramifying on the ventricular wall, such a mechanism has been discussed by Gordon^{25, 26} when he described erosion of the ventricular wall beneath the intraventricular blood. The subepen-

dymal vessels in the region were distended and some were also occluded by thrombus. Increased pressure within the subependymal vessels presumably resulted in distention with subsequent rupture and bleeding into the ventricle. Objective assessment of the pathogenic mechanism proposed by Gordon is complicated by the absence of histologic photographic documentation in these early papers. As well, multifocal erosion of the superficial ventricular ependymal lining may occur *secondary* to the presence of intraventricular blood, as described focally in the pathologic findings in our first case.

The neuropathologic evidence in our first patient clearly shows the IVH to be unrelated to any large intraparenchymal hematoma, berry aneurysm or vascular malformation (parenchymal or choroid plexus) that might have ruptured to produce IVH. One intriguing histopathologic finding is the presence of a small region of hemorrhagic necrosis in the wall at the angle of the left lateral ventricle. This is clearly in continuity with a large blood clot in the ventricular cavity. One of two scenarios to explain this finding could be envisioned. Either bleeding occurred into the ventricle for reasons that are not apparent, and blood then dissected into the adjacent brain parenchyma. However, the depth of tissue erosion is considerably greater than one usually observes with an intraventricular clot that has produced ependymal damage. Alternatively, there was a small hemorrhagic infarct in the parenchyma at the edge of the ventricle, which ruptured secondarily through the ependyma into the ventricular cavity to produce the large IVH illustrated in figure 1. We favour the latter explanation. The patient's moderately abnormal PT immediately prior to death may have contributed to the extent of the hemorrhage.

It is interesting to speculate on the possible relationship between the pathologic substrate of primary IVH, and that of hemorrhage into the caudate nucleus.²⁷ Hemorrhage into the latter structure can extend into the lateral ventricle, though the microscopic substrate of such hematomas has not been well documented.²⁷ Nevertheless, as with the examples of IVH we have described, the patients may have a relatively typical presentation that simulates subarachnoid hemorrhage and show good long term clinical recovery.

Intraventricular hemorrhage is common in premature infants. In that setting the hemorrhage usually originates in the periventricular germinal matrix and dissects into the ventricular cavities and (often) the subarachnoid space.^{28, 29} The precise pathogenic mechanisms involved in this type of perinatal hemorrhage are highly controversial, but probably involve some combination of abnormalities of cerebral autoregulation and blood flow, structural "weakness" of the germinal matrix vessels, systemic metabolic and coagulation abnormalities, as well as derangements of perivascular tissue pressure within the germinal matrix.²⁸⁻³⁰ Since all hypotheses concerning germinal matrix intraventricular hemorrhage in this age group take into account the unique anatomic features of the periventricular zone at this early stage of brain develop-

ment, they are extremely unlikely to have relevance to the adult situation. It is also accepted that in infants, the periventricular region is a watershed zone between the ventriculopetal and ventriculofugal branches of the deep penetrating arteries, and infarcts (periventricular leukomalacia) may also occur in this area.³¹ In the adult brain, the watershed regions, however, are commonly believed to occur between the major vascular territories (e.g. anterior-middle cerebral arteries) and watershed infarcts involve cortex and adjacent white matter rather than deep periventricular structures. Deep central grey matter, including periventricular structures, are the site of lacunar infarcts, the causes of which are themselves somewhat controversial.³²

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In accordance with *Stroke* policy, this article was guest edited by J.P. Mohr.

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The Sex Difference in Manifestations of Carotid Bifurcation Disease

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SUMMARY One would think that risk factors for transient ischemic attack (TIA) and asymptomatic carotid bruit (ACB) would be similar. In our referral population and in several previously reported cohort populations, however, men outnumber women among patients with TIA. In contrast, women outnumber men among patients with ACB. We found in two independent populations that women with ACB are up to 5.7 times less likely than men to have carotid stenosis. Thus women are more prone than men to have ACB, but their bruits much less commonly reflect carotid stenosis. Women are probably predisposed to have carotid bruit even in the absence of carotid stenosis. In our referral population of ACB, this tendency among women for carotid bruit without stenosis does not seem to be related to lower hematocrit, higher prevalence of heart murmur, constitutionally smaller carotid arteries, or differences in pulse rate or body habitus.

Stroke Vol 17, No 5, 1986

TRANSIENT ISCHEMIC ATTACKS (TIAs) are often caused by atherosclerosis of the carotid bifurcation. One would expect risk factors for TIA, asymptomatic carotid bruit (ACB), and asymptomatic carotid stenosis to be similar; however, in our Stroke Registry men outnumber women with TIA while women outnumber men with ACB. This observation prompted us to study our referral population to confirm this "gender gap" and investigate its cause.

Materials and Methods

Three separate, non-overlapping populations of patients were studied. Two were chosen from our Stroke

Registry, those with the diagnoses of ACB and TIA. A third population, composed of consecutive patients referred to our outpatient carotid ultrasound lab with the diagnosis of ACB, was employed to confirm findings in the Registry ACB population.

Our Stroke Registry is composed of data on all patients on the neurology inpatient service of the North Carolina Baptist Hospital with confirmed discharge diagnoses of TIA, ACB, subclavian steal syndrome, or transient global amnesia. Patients qualify for the diagnosis of TIA only if their histories conform to criteria defined by the Joint Committee for Stroke Facilities.¹³ Occasional patients with remote cerebral infarction and recent TIAs are included in the category of TIA. The category ACB is used in the Registry to denote a murmur localized over the mid-or-upper common carotid artery in patients without previous, ipsilateral, carotid ischemic symptoms. For this study, patients with ischemic symptoms in the distribution of the contralateral carotid artery or vertebral basilar circulation were also excluded. Patients with radiating heart murmurs, venous hums, and other non-carotid vascular sounds in the neck were not included. Those with

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