Article abstract—At least 20 different lacunar syndromes have been described and can be recognized by characteristic clinical features. Almost all occur in patients with hypertension. Small lacunes are usually due to lipohyalinosis, larger ones to atheromatous or embolic occlusion of a penetrating vessel. The concept of the "lacunar state" is examined in the light of recent knowledge with the conclusion that the clinical deficit is primarily related to unrecognized normal pressure hydrocephalus rather than to the presence of a few lacunes. The notion that lacunes occur haphazardly is criticized because the first or only lacune tends to be symptomatic. The incidence of cerebral lacunes has declined since the introduction of antihypertensive therapy, an indication that therapy is effective.

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Lacunar strokes and infarcts: A review

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Lacunar infarcts are small infarcts that lie in the deeper noncortical parts of the cerebrum and brainstem and result from occlusion of penetrating branches of the large cerebral arteries—middle cerebral, posterior cerebral, basilar, and less commonly, anterior cerebral and vertebral arteries.[1] An alternative terminology for this type of stroke would be "penetrating branch occlusion." In healing, the infarcts leave behind a small cavity, or a lacune, which is seen better in white matter structures than in gray matter, where some collapse of the cavity is the rule.

Small deep infarcts are not classed as lacunes if they are only part of larger infarcts that involve the cortex and are due to occlusion of large arteries. The term connotes a vascular origin and should not be used for other types of parenchymal lesions.

Lacunar infarcts range in size from large (1.5 to 2.0 cm) to very small (3 to 4 mm). The larger ones tend to be symptomatic, whereas the very small are liable to be asymptomatic unless strategically located in a sensory or motor tract. Pathologically, the most common lacunar sites are in approximately descending order of frequency: putamen, caudate, thalamus, pons, internal capsule, and convolutional white matter. In the symptomatic and therefore larger lacunes, the cause of the vascular occlusion is most frequently a tiny bead of atheroma in arteries that range in size from 400 to 900 μ.[2,3] Occlusion of a penetrating branch from the basilar artery often occurs at its origin if the mouth of the branch artery is blocked by an atheromatous plaque in the wall of the basilar artery itself.[3]

In pathologic studies the artery running to the region of a lacunar infarct may be normal throughout its length; by inference, we assume that a small *embolic particle* of thrombus from the heart or a parent large artery caused the obstruction,[2] because emboli generally undergo lysis. Small lacunar infarcts, 3 to 7 mm in diameter, most often are the result of occlusion by *lipohyalinosis*,[4,5] a hypertensive cerebral vasculopathy in which the lumen of an artery, usually less than 200 μ in diameter, is occluded, the wall of the artery is thinned and reduced to connective tissue shreds, hemosiderin-filled macrophages lie scattered in the vicinity, and the wall stains bright red with oil-red-O. This same type of lesion is probably the source of hypertensive hemorrhage. Rarely, dissection of a small artery, either at its origin or along its course, may cause a lacune.[2] In about 90% of cases, lacunar infarcts are associated with systemic arterial hypertension, defined as a blood pressure of greater than 140/90 mmHg.[6]

Clinical features. Lacunar strokes are often distinctive in their symptomatology, and a positive diagnosis can be made. Accuracy of clinical detail is an important requisite. Lacunes generally do not cause such deficits as aphasia, apractagnosia of the minor hemisphere, sensorimotor stroke, monoplegia, homonymous hemianopia, isolated severe memory impairment, stupor, coma, loss of consciousness, or seizures; the presence of any of these almost always excludes a lacunar diagnosis. Angiography usually does not disclose the occluded artery. CT detects the larger lacunes of the basal

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17. Lateral medullary syndrome
18. Lateral pontomedullary syndrome
19. Loss of memory (?)
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21. Miscellaneous:
    (a) Weakness of one leg with ease of falling
    (b) Pure dysarthria
    (c) Acute dystonia of thalamic origin

ganglia but not those in the thalamus and often not those in the pons. A knowledge of lacunar syndromes assists especially in clinicoradiologic correlation and helps prevent errors, both medical and surgical, which might otherwise be made in the patient's management.

The various syndromes proved pathologically or based only on clinicoradiologic experience are listed in the table.

1. Pure sensory stroke or transient ischemic attacks (TIAs).[7,8] This, the most common lacunar manifestation, takes the form of numbness of the face, arm, and leg on one side in the absence of weakness, homonymous hemianopia, aphasia, agnosia, and apraxia. The cases tend to fall into two almost mutually exclusive groups, one in which numbness is persistent from the onset, and the other in which it occurs in the form of TIAs; in only 10% of cases are TIAs the prodrome to persistent numbness. Although involvement of all three regions—face, arm, and leg—is usual, in two pathologically studied cases the numbness involved the face and hand in one and the mastoid region, arm, and leg in the other, indicating that all three regions need not be affected. Corresponding to the numbness, there may be a severe sensory deficit to all modalities or, on the other hand, little or no deficit. The infarct lies in the sensory (posteroventral) nucleus of the thalamus, and the underlying vascular disease is usually lipohyalinosis. CT has never demonstrated the lesion in personally studied cases.

Although the clinical details of the pure sensory syndrome are often straightforward, the number of varied patterns with which simple paresthesias resulting from a stroke can present is surprising. In a recent survey[9] 135 personally studied patients with heminumbness who were candidates for the diagnosis of pure sensory stroke were classified into six categories as follows: pure sensory stroke, 58 cases; pure sensory TIAs, 42 cases; atypical hemiparesthesia, 20 cases; hemidysesthesia, 9 cases; cervical disk disease suspect, 5 cases; and 1 special case of thrombocythemia. "Numbness" was by far the most common term used by these patients in describing their main symptom. Post-stroke thalamic pain occurred in only three cases. In some 16 cases there arose the possibility of late-life migrainous accompaniments,[10] and, as usual, lack of reliable criteria prevented a diagnostic decision. The atypical cases constituted a most heterogeneous collection that defied simple classification.

Since the diagnosis of pure sensory stroke rests wholly on the clinical findings, it was of interest to see whether the numbness of strokes associated with occlusion of the posterior cerebral and internal carotid arteries mimicked that of pure sensory stroke. In 70 cases of occlusion of the posterior cerebral artery, prodromal TIAs of numbness closely resembled pure sensory TIAs in 7 cases (10%), whereas in 100 cases of occlusion of the internal carotid artery, there was a resemblance in only one case.

2. Pure motor hemiparesis.[2,12] This is the second most frequent syndrome and refers to a pure motor stroke involving face, arm, and leg on one side in the absence of sensory deficit, homonymous hemianopia, aphasia, agnosia, and apraxia. This definition always refers to the acute stage of the stroke. The rules apply only when face, arm, and leg are involved; pathologic verification has not been obtained in a case with involvement of only face and hand, or hand and leg. Monoplegia is never due to occlusion of a penetrating branch. At the onset of the stroke there may be minor numbness of one or other region, usually the face or a few fingers, but no sensory deficit is found on examination. In one of our cases with persistent numbness, CT showed an unusually large capsular lesion suspected of being the result of embolism. Although there is almost never a headache at the onset, about 1 in 20 of the basilar branch patients has head pain, the mechanism of which is not known. Two patients have had asterixis of the involved limbs.

The stroke may develop on a background of pure motor hemiparesis (PMH)-TIAs, it may come on during sleep, or it may develop in a stepwise fashion over 2 to 6 days or evolve gradually in 3 days. The tendon reflexes in the affected limb become brisker in the first hours of the stroke. The infarct is located

in the posterior limb of the internal capsule in the region of the junction of the anterior two-thirds and posterior one-third, in the lower basis pontis where the corticospinal tracts are congregating to form the pyramids, or rarely in the midportion of the cerebral peduncle. CT will demonstrate the larger lesions of the internal capsule, putamen, and caudate nucleus, but may fail to detect lesions of the lower pons or smaller lesions of the internal capsule. CT evidence of a lacune becomes less distinct with the passage of time.

The vascular disease process lies in a branch arising from the middle cerebral, lower basilar, or upper vertebral artery and takes the form of atherosclerosis of the branch, occlusion of the mouth of the branch by a plaque in the parent artery, or embolism. In addition, the lesion in the internal capsule may be the result of either lipohyalinosis or dissection of a penetrating artery.[2] Occlusion or stenosis of the internal carotid artery has not been associated with pure motor hemiparesis (PMH) in personally studied cases, but a small embolic particle could lodge in the appropriate penetrating branch. Rarely, stenosis of the stem of the middle cerebral artery will block the mouth of a penetrating artery, causing PMH combined with prominent abulia.

3. Ataxic hemiparesis.[12,13] This form of stroke is the result of infarction of the basis pontis at the junction of the upper one-third and inferior two-thirds. PMH on the opposite side is combined with cerebellar dysmetria in the affected limbs, provided that there is sufficient power to carry out the tests. The leg may be more affected than the arm, and the face least of all. In the upper basis pontis, the corticospinal fibers (running from the cerebral peduncle to the pyramid) are dispersed into bundles by the nuclei pontis, thereby providing the basis for selective involvement of individual groups of fibers, for example, the arm or leg predominantly. By contrast, in the cerebral peduncle and pyramid, the fibers for all three regions (face, arm, and leg) form a compact bundle, and involvement of individual regions will be unlikely. The type of ataxia referred to here is not caused by the weakness of a limb but should always suggest a pontine lesion. There may be dysarthria, nystagmus, and toppling to one side. At the onset there often is numbness of the face or face and hand or occasionally the foot, without demonstrable sensory deficit, no doubt reflecting involvement of the medial lemniscus. Occasionally the foot and ankle are selectively weak, resulting in the picture of foot drop combined with cerebellar ataxia, increased tendon reflexes and a Babinski sign. In one case, the affected foot stayed "glued" to the floor while the patient circled around it with the other leg; when the patient was lying down, weakness of the leg was not prominent. CT usually fails to show the lesion. The infarct is due to occlusion of a paramedian branch of the basilar artery by atheroma in the branch or parent artery or by embolism.

4. Dysarthria-clumsy hand syndrome.[14] Here facial weakness, severe dysarthria, and dysphagia are combined with slight weakness and clumsiness of the hand (noticeable, for example, in writing), brisk tendon reflexes, and a Babinski sign on the same side. The responsible lesion is either in the basis pontis at the junction of the upper one-third and lower two-thirds or, as happened in one case, at the genu of the internal capsule in its uppermost part. There is no sensory deficit. When a closely similar syndrome is the result of a superficial cortical infarct, careful examination will show a sensory deficit on the affected lips. It is probably best to regard this syndrome as a variant of number 3.

5. Modified PMH with "motor aphasia." In the only pathologically studied case, a large softening involved the genu and anterior limb of the internal capsule and the adjacent white matter of the inferior corona radiata. Thrombotic occlusion of a lenticulostriate branch supplying the territory of the infarct was identified. The patient showed severe weakness of the right side of the face and moderate weakness of the right hand. The right leg was slightly weak, and there was a Babinski sign. Early in the illness there was dysarthria, and the patient mispronounced words but corrected the errors. Later he made single-syllable unintelligible sounds and finally was mute. Comprehension remained good, but responses were long delayed.

6. PMH sparing the face.[15] This is caused by an infarct of the medullary pyramid because of occlusion of either the vertebral artery itself or a penetrating branch therefrom. At the onset there may be slight vertigo and a trace of nystagmus that suggest the correct localization. Should there be prominent numbness or involvement of the tongue, the clinical picture is that of the medial medullary syndrome. The other pyramid may become similarly involved at a later date, producing quadriplegia.

7. Mesencephalothalamic syndrome—third-nerve palsy, paresis of vertical gaze, and abulia. There are several variations of this syndrome, which is the result of occlusion of one or more of the arteries of Percheron[16]—anterior thalamosubthalamic paramedian arteries, posterior thalamosubthalamic paramedian arteries, and the superior and inferior mesencephalic paramedian arteries, all of which arise from the proximal 3 or 4 mm of each posterior cerebral artery. The infarct typically has a butterfly shape and involves the paramedian regions of the midbrain, subthalamus, and thalamus bilaterally. Bilateral lesions are the result of the anatomic arrangement; a penetrating branch from one posterior cerebral artery may supply both sides of the midline in midbrain, subthalamus,

and thalamus. There may be unilateral or bilateral third-nerve palsies, Parinaud syndrome, or selective paralysis of downward gaze that are often combined with drowsiness, abulia, and impairment of memory.[17] Judging from personal experience, the syndrome is embolic in 90% of cases. On the Stroke Service of the Massachusetts General Hospital, it has gained the colloquial designation "top o' the basilar embolism." The tendency for emboli to enter these small vessels is probably the result of their anatomic position, directly in line with basilar flow. Prodromal symptoms are usually lacking. When occlusion of the basilar or posterior cerebral artery is responsible, involvement of neighboring territory will provide a clue. Usually CT does not disclose this infarct.

*8. Thalamic dementia.*This is a special variation of number 6 in which the central region of one thalamus and the adjacent subthalamus are affected as a result of entrance of an embolus into an anterior thalamosubthalamic paramedian branch, which atypically has a unilateral distribution. Neurologic examination will disclose abulia, impairment of memory, and a partial Horner syndrome (miosis and anhydrosis). CT may well show the infarct.

9. PMH with horizontal gaze palsy.[3] In the only pathologically proved case, a paramedian infarct in the lower pons caused PMH affecting face, arm, and leg combined with a transient one-and-a-half syndrome (ipsilateral conjugate lateral gaze palsy and crossed internuclear ophthalmoplegia), leaving abduction of the opposite eye as the only horizontal eye movement, reflecting preservation of the opposite sixth nerve.

10. PMH with crossed third-nerve palsy (Weber syndrome). This is a rare event in which an infarct involving the midportion of the cerebral peduncle catches the issuing fibers of the third nerve.

11. PMH with crossed sixth-nerve palsy. Here an infarct involves the paramedian inferiormost pons, catching the fibers of the ipsilateral sixth nerve as they exit from the brainstem.

12. PMH with confusion. In a single pathologically studied case, a left PMH was associated with the acute onset of confusion and impairment of attention and memory. The only lesion in the brain was a 1.2-cm lacune in the anterior limb and anterior part of the posterior limb of the right internal capsule. Ostensibly the thalalmofrontal connections were interrupted. Confusion is rarely a lacunar manifestation.

13. Cerebellar ataxia and crossed third-nerve palsy (Claude syndrome). In this syndrome, a small infarct in the dentatorubral tract (superior cerebellar peduncle), after it has crossed the midline, also affects the intramesencephalic fibers of the third nerve.

14. Sensorimotor stroke (thalamocapsular).[18] In the only pathologic study of such a case, an infarct in the posterolateral thalamus involved the sensory nucleus and extended into the adjacent posterior limb of the internal capsule. The stroke at the onset was pure sensory in type, but later a hemiparesis of moderate degree was added; this subsequently resolved, leaving a residual pure sensory stroke. The underlying vascular process was lipohyalinosis.

15. Hemiballism. Classically, an infarct (or hemorrhage) in the subthalamic semilunar nucleus of Luys is the cause of hemiballism. The vascular lesion has not been traced out, and it is uncertain whether the occluded arterial branch arises from the posterior cerebral, posterior communicating, or anterior choroidal artery.

16. Lower basilar branch syndrome. Satisfactory pathologic study has not been possible in these important cases, which are frequently puzzling. Included here are strokes characterized by the following disturbances, occurring either singly or in combinations in the absence of significant occlusive vascular disease in the lower basilar artery on angiography: dizziness (vertigo), nystagmus, diplopia, horizontal gaze palsy, internuclear ophthalmoplegia, dysphagia, appendicular ataxia (cerebellar), overbalancing to one side, staggering, facial weakness, burning feeling in the eyes, and trigeminal numbness. It is postulated that the responsible occlusion lies in a small branch arising from the lower basilar or upper vertebral artery that supplies the tegmentum of the lower brainstem without involving the corticospinal system. Thrombosis, embolism, and mural plaque in the parent artery are possible mechanisms. Ectasia of the basilar and vertebral arteries may be a predisposing factor. Partial syndromes resulting from occlusion of a vertebral, posterior inferior cerebellar, anterior inferior cerebellar, or superior cerebellar artery should be excluded. There have been a few cases in which, after satisfactory recovery from the initial stroke, an almost identical stroke occurs 1 to 5 years later.

17. Lateral medullary syndrome. This syndrome, which is usually the result of occlusion of a vertebral or posterior inferior cerebellar artery, may result from occlusion of a small branch of a vertebral artery. However, a detailed microscopic examination has not been made.

18. Lateral pontomedullary syndrome. The few pathologic studies of this syndrome have demonstrated occlusion of a small aberrant arterial branch arising from the upper vertebral artery and running superiorly and laterally to the region of exit of the seventh and eighth nerves, a veritable short circumferential artery. The clinical picture includes vertigo, vomiting, facial weakness, dysarthria, dysphagia, tinnitus, cerebellar ataxia, nystagmus, ipsilateral Horner syndrome, ispilateral trigeminal numbness, and sensory deficit and spinothalamic sensory loss on the opposite side of the body.

19. Loss of memory. A single pathologic study disclosed a lacunar infarct in the corpus callosum with involvement of the pillars of the fornix immediately subjacent. No other cause was found for the patient's sudden onset of impairment of memory.

20. Locked-in syndrome.[19,20] This is a result of bilateral pure motor hemiplegia resulting from infarction of the corticospinal tracts bilaterally at any of the appropriate levels—internal capsule, pons, pyramid, or, rarely, cerebral peduncle.

21. Miscellaneous. (a) Weakness of one leg with ease of falling. In this pathologically unverified syndrome, a hypertensive patient will note slight weakness of one leg or of the leg and arm, combined with a remarkable tendency to fall toward the weak side in the absence of an adequate explanation. Angiography is normal. A brainstem location is suspected. (b) Pure dysarthria. This clinical picture, which has been encountered several times in hypertensive patients, lacks pathologic verification. The sudden onset of unaccompanied dysarthria points to a vascular insult, but angiography and CT are normal. A location in the basis pontis is favored. (c) Acute dystonia of thalamic origin.

Asymptomatic lacunes. Unless they extend to the adjacent internal capsule, lacunes in the head of the caudate nucleus and in the putamen are silent; at least no specific symptoms have been recognized. There is no convincing evidence that the entity "arteriosclerotic parkinsonism" exists. Small lacunes in the cortical white matter and in the central part of the thalamus are usually asymptomatic.

Management. The management of patients with a lacunar stroke will depend on the presumed nature of the vascular disease, and generalization is not warranted. In pure sensory stroke the vascular process is lipohyalinosis, which typically is associated with hemosiderin-filled macrophages, indicating slight extravasation of erythrocytes. Hence, heparin and sodium warfarin are contraindicated, but aspirin and dipyridamole may be tried. In PMH the vascular disorder is less likely to be lipohyalinosis, and, therefore, treatment with heparin and sodium warfarin may be undertaken if the patient when first seen is in the stage of multiple TIAs of PMH or is in the group with stepwise or gradually progressive hemiparesis. Personal experience indicates that in this situation anticoagulant therapy will prevent or attenuate the stroke. In the absence of prodromal TIAs, exclusion of a hemorrhage by CT is mandatory. When an embolic cause is suspected, attention is directed to the prevention of recurrence.

In general, the prognosis for recovery from a lacunar deficit is excellent, and a prolonged rehabilitation effort will be rewarding. Occasionally a PMH fails to follow the rule, and in some of these cases there will have been previous damage to the contralateral corticospinal system.

L'État lacunaire: The lacunar state. Marie[21] was the first to describe *l'état lacunaire*, or the lacunar state, in which the presence of one or more lacunes was associated with a chronic progressive neurologic decline often marked by one or more episodes of slight hemiparesis and ending with invalidism, dysarthria, a small-step gait (marche à petits pas), imbalance, incontinence, pseudobulbar signs, and some degree of dementia. Bilateral spastic hemiparesis was never part of the picture. Von Malaisé[22] among others was not satisfied with the clinicopathologic correlation and pointed out that the hemiparesis was minor and that the leg was more involved than the arm, just the reverse of most cases of hemiparesis. He noted that in some patients who had had a stroke involving only one side, function of both legs was disturbed, and, furthermore, some patients had never had a stroke. Marche à petits pas was present whether the internal capsule was involved or not, and patients tended to be abulic and if forced to bed by an intercurrent illness were liable never to walk again. Ferrand[23] found 217 lacunes in 88 cases examined pathologically, an average of less than 3 lacunes per patient, a small number considering the reduced neurologic state of these patients.

Judging from personal experience, one rarely (if ever) encounters the lacunar clinical state as described above. In at least some of Marie's[21] patients, much of the clinical picture was probably due to unrecognized symptomatic normal-pressure hydrocephalus. In his cases hydrocephalus was usually prominent pathologically, and illustrations show an "enormous" ventricular dilatation. Earnest et al[24] and Koto et al[25] have reported the association of lacunar infarcts and normal-pressure hydrocephalus. Normal-pressure hydrocephalus, by its compressive effect, may predispose to lacunes in the cortical white matter.

A general impression prevails that multiple lacunes are distributed haphazardly, some symptomatic, some not, with the accumulation of lesions gradually producing a complex picture of neurologic deficits. The findings in a pathologic study of nine cases of lacunar infarction of the internal capsule associated with PMH,[2] however, were contrary to that impression, as they clearly indicated a tendency for the first lacunar infarcts to be symptomatic. In five cases the capsular infarct causing PMH was the only lesion in the brain. In two further cases, there were two lacunes. In the first of these, both were capsular infarcts, and in the second case, one was capsular and one was a thalamic lacune associated with a pure sensory stroke. Two patients had three lacunes; in one patient, two were capsular, and in the other patient, one

lacune was capsular. In two additional cases, one had a single lacune that was capsular and one had four lacunes, two of which were capsular. Of a total of 21 lacunes, 15 were capsular and were associated with PMH. In these cases survival for several years after the stroke was not unusual, providing ample time for other lacunar lesions to have occurred.

To investigate this matter further, other cases with lacunar strokes were reviewed. Of three cases of pure sensory stroke studied pathologically,[8] two had a thalamic lacune as the only lesion in the brain, whereas one had two lacunes, one in the thalamus and one in the internal capsule. In the thalamocapsular case referred to in Number 14 in the lacunar syndromes, there was a single lesion. Of three cases of ataxic hemiparesis, two patients had a single lacune and one patient had two lacunes. Of the total of 18 patients, in 13 the only lacune or the first lacune was symptomatic.

Clinical experience is in agreement. In a personal series of 47 cases of PMH (most studied only clinically), the lacunar stroke was always the first stroke. In 135 cases of pure sensory stroke, almost all studied only clinically, the pure sensory stroke was the first manifestation of cerebrovascular disease in 120 cases.

These data support the proposition that small penetrating branch artery disease tends to occur where it gives rise to symptoms rather than being distributed by chance. The notion of numerous asymptomatic lacunes lying scattered at random is probably wrong. In clinical deliberations, the concept that several lacunes may accrue silently should be entertained only with reservation. The explanation for this predilection is not obvious. Perhaps, blood flow to the motor and sensory tracts has special characteristics; for example, total volume or fluctuation may predispose these arteries to occlusive disease.

Is the incidence of lacunar infarcts decreasing?

In a pathologic study of lacunes spanning the years 1950 to 1954[1] 114 brains (11%) out of 1042 disclosed lacunes. Fifty-four brains (47.7%) contained one or two lacunes, and 60 (52.6%) contained three lacunes or more; there were 3 lacunes in 17 cases, 4 in 12, 5 in 4, 6 to 10 in 18, and more than 10 in 9, making a total of 376 lacunes, or 3.3 per brain. In a comparable retrospective study of 200 brains examined in the years 1975 and 1976, 16 (8%) disclosed one lacune or more. Thirteen (81%) contained 1 or 2 lacunes, and 3 (19%) contained 3 or more; 3 in 1, 5 in 1, and 7 in 1, making a total of 33 or 2.5 per case. From neuropathologic observations, one gets the impression that the florid lacunar states that were common in the years before the use of antihypertensive therapy are becoming almost a rarity, and the above data tend to confirm this, although it cannot be claimed that the study would bear rigid statistical scrutiny. These results support the widely accepted view that antihypertensive therapy is proving effective.

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