SUMMARY  Pure motor hemiplegia (PMH) is a well defined syndrome usually caused by ischemic lesions of lacunar type located either in the internal capsule or in the pons. Angiography and isotope scanning are usually normal. CT scan reveals small deep infarcts and appears to be the most reliable investigative method. The CT scan findings are described of thirty patients with PMH of rapid onset (less than 36 hours). In 29 of the 30 cases a lesion was found which could explain the PMH. Small hemorrhages (2 cm in diameter) in the posterior limb of the internal capsule were noted in two cases. Ischemic lesions were found in 27 patients, 22 patients had a single lesion (20 capsular and 2 pontine), while 5 patients had 2 lesions (2 bi-capsular, 3 capsular and pontine). Three varieties of ischemic capsular lesions were observed. We found in 15 cases a capsulo-pallidal infarct (type I); in 8 cases a capsulo-pallidal infarct (type II); and in 2 cases an anterior capsulo-caudate infarct (type III). Type I corresponded to the territory of the internal lenticulostriate branches of the anterior cerebral artery. Type II involved the territory of the perforating branches of the anterior choroidal artery. We suggest that type III involves the territory of the internal lenticulostriate branches of the anterior cerebral artery. Lacunes are generally linked to arterial systemic hypertension. However, only 16 of 30 patients in this series were chronically hypertensive.

AS early as the turn of the 20th century, P. Marie and Ferrand demonstrated that lacunes in the internal capsule may be responsible for an isolated hemiparesis. In 1965, a study published by Fisher and Curry outlined concisely the clinical criteria of Pure Motor Hemiplegia (PMH); complete or incomplete paralysis of the face, arm, and leg without loss of sensation or disturbance of visual field, dysphasia, apraxia or agnosia. These criteria apply to the initial phase of the syndrome, excluding cases in which the symptoms are present at the onset but regress secondarily. According to these authors, the syndrome might have been caused by lacunar type ischemic lesions located either in the internal capsule or the pons. Subsequently, several isolated cases were published revealing other possible locations for the lesions: infarction in the region of the Rolando fissure and infarction in the region of the anterior cerebral artery, and lacunes in the brainstem involving the decussation of the pyramids. In addition, other etiologies of PMH have been described and include hemorrhage, metastasis and abscess.

PMH constitutes a neurological syndrome which has not yet been fully understood. There are a few clinical pathological reports. Angiography and isotope scanning are usually normal because of the small size of the lesions. However, CT scanning can show the largest lacunar lesions. The purpose of this study is to describe the CT scan finding in thirty patients with PMH.
Patients and Methods

Thirty patients with PMH were included in this study. The onset of the symptoms in all patients occurred in less than 36 hours. All of them were examined by one of us before the end of the third day. The age, sex, vascular risk factors, side of involvement and characteristics of the hemiplegia are given in tables 1 and 2. The onset and progression of the PMH were variable. In 14 patients there was immediate complete deficit, eight of these with earlier preceding TIA's (2 to 8 incidents) manifest by repetitive hemiplegia in the week preceding the definitive accident. Two patients had steadily progressive deficit over a course of several hours. In 14 patients step-by-step deficits occurred.

Besides the motor deficit, we observed as did Fisher and Curry, transient neurologic signs during the first few days: dysarthria not explained by facial paresis in 5 patients, dysphonia and difficulty in swallowing in 3 patients, problems in conjugate gaze towards the side of hemiplegia in 2, in 4 patients there were complaints of "dizziness" during the first hours. All of these signs regressed in less than 48 hours.

Laboratory findings included a normal CSF in all cases. The EEG showed an area of slow activity on the side of the lesions in 3 patients. Isotope studies were performed in 15 patients. Small zones of radionuclide uptake on the same side as the lesion were noted in 3. Angiography was performed in 21; in 9 patients vertebral and carotid angiography was done via the femoral route with magnification in 4 of them to improve the visualization of perforating arteries. All angiograms were performed within 3 weeks of the ictus. In 12 cases, intravenous angiography of the great vessels of the neck was performed. No carotid or vertebral stenosis were found.

The follow-up was longer than three months in 25 patients. In 19 of these, the clinical outcome was favorable leaving only a minor motor deficit. In 6 patients, there were major sequelae involving functional incapacity of the upper extremity. All patients, however, recovered the ability to walk.

Computerized Tomography

A Delta-25 head scanner was used. In all cases 8 mm slices were performed parallel to the canthomeatal line. The internal capsule (IC) can be visualized in 4 slices. Examination of the posterior fossa, with close attention to the region of the cerebellum, pons, and medulla, was included in all cases. In each patient, CT was performed before and after intravenous injection of contrast medium (30% meglumine ioxithalamate 2 ml/Kg). In 27, the examination was performed between the 8th and 30th day after the onset of symptoms. In one, it was performed during the 2nd day and in 2 after 90 days. A follow-up CT study was performed between the 41st and 180th day in four patients.

Results

In 29 of the 30 patients, a lesion was found which could explain the PMH (table 3). In 2, hemorrhage was observed in the posterior limb of the internal capsule before injection of contrast. This appeared as a zone of hyperdensity outlined by a thin rim of hypodensity (fig. 1). Ischemic lesions were found in 27 patients: 22 patients had a single lesion (20 capsular and 2 pontine) while 5 patients had 2 lesions. In the 5 cases with multiple lesions, there was always a capsular lesion contralateral to the PMH. The second lesion had a variable topography. In 2
patients, a lesion was identified in the ipsilateral internal capsule. In 3 patients, the second lesion was pontine, 2 ipsilateral to the PMH, and 1 contralateral to the PMH. In this last one it was difficult to discern which of the 2 lesions was directly responsible for the hemiplegia; neither of the 2 lesions enhanced after contrast medium injection.

The pontine lesions observed were zones of low absorption values approximately 8 mm in diameter located in the anterior contralateral pons (fig. 2). In 2 cases with a single pontine lesion on the appropriate side, patients described a sensation of dizziness without vestibular signs. There was no evidence of conjugate gaze disorder. In the only case in which the clinical course was well established, partial regression was noted.

Three different capsular lesions were found and could be classified depending on their location and morphology:

**Type 1: Capsulo-putamino-caudate infarcts (15 patients)**

In 4 patients, the hypodensity was widespread and was observed on all 4 CT slices showing the internal capsule (fig. 3, 4). The lesion extended from the anterior limb of the IC posteriorly through the putamen to the posterior limb of the IC and from the inferior aspect of the putamen up to the corona radiate and body of the caudate nucleus. The zone of hypodensity was semi-lunar in shape with its medial aspect concave to the midline. The superior portion was medial to the inferior portion. In these patients hemiplegia was massive and affected arm and leg equally (proportional). There were major sequelae in 3 cases; the fourth concerned an 8 year old child with faciobrachial hemiplegia and complete recovery.

In 11 patients the lesion was smaller and could only be seen on the three highest slices, but retained the same general shape. Unlike the preceding cases, the interior aspect of the putamen was spared. The hemiplegia was proportional in 5, faciobrachial in 5, and crural in one. Nine of these patients were followed: 7 had a complete remission; 2 of those with initial proportional hemiplegia showed signs of continuing spastic weakness.

In 6 of these 15 patients, cerebral angiography was performed. In 3 it was normal; in 1 a delayed passage of contrast was noted in the lenticulostriate arteries without visible occlusion; 2 exhibited occlusion of the external lenticulostriate arteries.

**Type 2: Capsulo-pallidal infarcts (8 patients)**

A zone of hypodensity was found in the posterior limb of the internal capsule (fig. 5); in some cases the internal portion of the globus pallidus was involved. The hypodensity never extended upwards to the corona radiata. In all cases, the lesion could be seen on only one slice. In the 7 patients in which a capsulo-pallidal lacune was isolated, the hemiplegia was proportional in three and was predominantly faciobrachial in four. In the 6 patients with adequate follow-up the clinical course was favorable. Angiography was performed in only one case and no lesion was demonstrated.

**Type 3: Anterior capsulo-caudate infarcts (2 patients)**

In the first patient, the lesion was found in the three superior CT sections of the internal capsule. The lesion was comma-shaped with its head located in the caudate nucleus and the tapered portion passing laterally and posteriorly forming a medial concavity, ending at the lateral border of the putamen (fig. 6). In the second patient, the lesion was smaller and remained in the region of the anterior limb of the cap-
sule and the head of the caudate nucleus. In both patients, the hemiplegia was predominantly facio-brachial, predominantly proximal in the upper limb in the first patient. Angiography was normal in both of them and recovery was complete in several days.

Of 27 patients with evidence of ischemia in the CT scans 24 patients were submitted to these studies between the 8th and 30th day following the onset of the deficit. Contrast enhancement was seen in only 13 cases (1 pontine lesion and 12 capsular). The presence of contrast enhancement appeared to be a function of lesion size.

Discussion

In this series of 30 patients with PMH the CT was normal in only 1 patient. In Weisberg's study of 33 patients with PMH, 9 had normal CT scans. In Nelson's study of 26 patients with PMH, there were 10 normal CT scans. The discrepancy may be related to the differences in technique used. Weisberg believed that a collimation of 8 mm would reduce the number of false negative examinations by detecting smaller lesions. The present study supports this view because capsulo-pallidal lesions were visible on only one CT slice with a collimation of 8 mm.

In our 29 cases with positive CT scans, the etiology was always vascular, 27 with evidence of ischemia, 2 with hemorrhage. Other causes have been reported in the literature. Weisberg's series included one with metastasis and 2 with multiple sclerosis; his study included patients in whom the PMH manifested itself over a period of days or weeks whereas the present report concerns patients with the acute onset of PMH.

A hemorrhagic origin of PMH is considered classically to be rare. Fisher and Curry did not find any case of hemorrhage in their series. However, they did not exclude the existence of hemorrhage as a cause of PMH. Igapashi reported a case of PMH secondary to post-operative hemorrhage. In our 2 patients, as well as Weisberg's, the lesions were small in size (less than 20 mm), and were located in the posterior limb of the internal capsule. No particular clinical finding or diagnostic procedure other than CT is able to differentiate hemorrhagic from ischemic PMH. This study confirms again the utility of CT in diagnosing intraparenchymal cerebral hemorrhage of small or medium size.

Ischemia is by far the most frequent finding but the topography of the infarcts is variable. In 9 autopsy-proven cases, Fisher and Curry found that 6 lesions were capsular and 3 pontine. Other locations have been described in occasional clinico-pathological
cases. In Weisberg’s CT scan study there were 6 cases with superficial infarcts. In 16 PMH patients evaluated with CT scan, Nelson found 11 small deep lesions, and 5 large superficial infarcts. In our 27 patients, the lesions were either capsular or infrequently pontine in location. In 5 CT scans, hypodense zones were located in the pons; but only 2 of these lesions could be considered responsible for the PMH. This lesion is topographically analogous to infarction of the pons described by Fisher and Curry and corresponds with the paramedian infarction of Foix and Hillemand. Fisher showed that these infarctions were most likely due to atheroma in the perforating branches of the basilar artery.

Three types of ischemic capsular lesions were observed:

Type 1

Type 1 corresponded to a capsulo-putamino-caudate infarction, identical to those lesions described by Fisher as “giant lacunes” and similar to the deep partial capsulo-putamino-caudate infarction of Foix and Levy. The infarction involved the territory of the lateral lenticulostriate branches of the middle cerebral artery. The arterial occlusion is rarely visualized angiographically and was proven in only 2 of 6 cases.

Type 2

Type 2 was a capsulo-pallidal infarction. This type corresponds to the capsulo-pallidal lacunes of Fisher and Curry. Foix and Levy described an identical lesion and noted that the middle cerebral blood supply in this region was relatively insignificant. Fisher suggested that an occlusion of the perforating branches of the anterior choroidal artery, which usually supplies the posterior limb of the internal cap-

Type 3

Type 3 corresponded to the capsulo-putamino-caudate and capsulo-pallidal lacunes of Fisher and Curry. Atheroma in the perforating branches of the anterior choroidal artery may be the causative factor.
sule, may have been responsible for this lesion. The small diameter of these vessels did not permit their visualization by angiography despite magnification.

Type 3

Type 3 was characterized by an infarction in the anterior limb of the internal capsule and in the head of the caudate nucleus. Fisher and Curry did not mention this type of lesion as a possible cause of PMH. The territory involved corresponds to the region supplied by the internal lenticulostriate arteries including the recurrent artery of Heubner, a branch of the anterior cerebral artery. Isolated occlusion of this artery is not well known. Foix and Levy reported the existence of small infarctions, involving the anterior limb of the internal capsule, which they attributed to occlusion of the anterior cerebral artery rather than to the middle cerebral artery. They admitted that their material was limited and that definite conclusions could not be drawn. Weisberg reported 2 cases of PMH due to an infarction of the anterior cerebral artery territory but the topography did not correspond with those observed in our type 3 lesions. Critchley wrote that “the involvement of the anterior portion of the internal capsule can result in severe facial and proximal upper extremity paralysis.” In the 2 patients of this series, the hemiplegia was predominantly facio-brachial; in one, the brachial deficit was clearly proximal; both subjects were right-handed and even though the lesions were on the left side no other neurologic manifestation (notably aphasia or apraxia) were observed. In 2 patients, angiography did not reveal any abnormality of the middle striate arteries. A possible explanation for PMH in type 3 infarction is suggested by a recent study by Ross. He stated that the pyramidal tract penetrates the internal capsule at the level of the anterior aspect of the posterior limb and then descends towards the posterior portion of the same limb. The existence of reversible ischemia at the periphery of a hypodense zone might explain the onset and complete rapid remission in approximately one week. In one of our 2 patients, a follow-up CT scan performed 3 months later revealed a decrease in the size of the lesion with sparing of the inferior aspect of the head of the caudate nucleus and an associated dilatation of the frontal horn of the lateral ventricle. The clinical course in cases of capsular PMH is not always favorable. In our study, recovery for type 2 and 3 lesions was good with few functional sequelae. This was not true for type 1 lesions where the degree of recovery was related to the size of infarction. Our observations, similar to those of Pullicino, suggest that the clinical variations and degree of recovery can be explained by differences in size and location of the infarction.

As Fisher reported, we noted certain transient neurological symptoms in the initial phase of the ischemic PMH: difficulty in swallowing in 3 cases (2 capsular, 1 pontine), severe dysarthria not explained by facial paresis alone in 5 cases (3 capsular type 1, 2 capsular type 2). Are these cases typically PMH syndromes? Nelson excluded from the syndrome patients with speech disturbances because he thought it was difficult to distinguish minimal dysphasic and non-dysphasic disturbances. All our patients with dysarthria were tested during the first days for specific aphasia screening: none were dysphasic. The dysarthria spontaneously regressed in several days. Unfortunately, a complete phonologic examination was not carried out, and as in the cases reported by P. Marie and by Fisher, its mechanism remains unknown. Proof of a bilateral lesion was found in only 2 of our 3 patients. They had Babinski’s sign contralateral to the hemiplegia. One patient had CT demonstration of a second lacune in the corona radiata opposite to the side of the capsular lesion responsible for the PMH. Fisher suggested that the cortico-bulbar fibers responsible for articulation are located in the genu of the internal capsule. In this perspective, a 58 year old patient, not included in this study, was admitted to hospital because of dysarthria and transitory acute confusion without a motor deficit. The CT scan revealed an isolated ischemic lacunar lesion in the genu of the left internal capsule. This case is very similar to cases 7 and 8 reported by Fisher.

It seems reasonable to think that further studies will reveal additional capsular syndromes. PMH is not the only syndrome due to capsular lesions. Nelson found one smaller capsular infarct in one patient with pure sensory stroke, and the capsular lacunae in 6 patients with symptoms compatible with the “dysarthria-clumsy hand syndrome.” Lesions located in the capsule may also be asymptomatic. The discovery of asymptomatic lacunes in the internal capsule is well known in neuropathology. Weisberg noted 5 asymptomatic capsular lesions in 8000 CT scans.

Capsular infarctions and lacunes are generally linked to the occlusion of perforating arteries of small caliber. Fisher described this as a “segmental arterial disorganization” in chronic hypertension, even when only moderate in severity. The present series lead us to question this hypothesis. An embolic mechanism is possible. This may have been also the case in 2 of our patients with chronic arterial fibrillation. In the others, 16 had no prior history of blood pressure exceeding 150/90 mm Hg; 19 patients were over 60 years of age; among the 11 patients under 60, only 4 had hypertension. This leads us to believe that hypertension is not always a prerequisite for arteriolar lesions which later might create lacunes.

References
PURPOSE OF PRIMATE FOCAL CEREBRAL ISCHEMIA BY INDOMETHACIN

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SUMMARY The effect of indomethacin (3mg/kg IA) preloading on the pathophysiology of a model of acute cerebral ischemia has been tested. Primates anaesthetised with alpha-chloralose were used. Indomethacin reduced basal blood flow by 39% and reduced CO2 reactivity by 71%. Water content changes of the cerebral cortex and relationships between blood flow and extracellular potassium (K), and calcium (Ca) activities have been measured. Indomethacin infusion did not affect the water content of the left side but there was more water in all regions of the right hemisphere which were rendered ischemic. These water increases were significant for blood flows greater than 5ml/100g/min in exposed areas. There was a significant increase in the flow thresholds for change in K and Ca. Possible mechanisms for these changes have been discussed.

Stroke, Vol. 13, No 1, 1982

IN primate cerebral cortex subjected to 30–90 mins of partial ischemia a significant increase in water content occurs in regions where blood flow is reduced to below 20ml/100g/min. In the same preparation, it has been shown that there is a critical level of blood flow around 10ml/100g/min below which the homeostasis of extracellular potassium (K) and extracellular calcium (Ca) activity is disrupted.

Increased outflow of prostaglandins from anoxic and ischemic brain is well known and suggests that prostaglandins are involved in the pathophysiology of ischemic cell damage. Indomethacin, a well known and potent inhibitor of prostaglandin synthesis, when given pre-insult has been shown to aid post-ischemic reperfusion, and to improve the quality of recovery of gerbils subjected to transient bilateral common carotid occlusion. These observations support the proposal that metabolites of arachidonic acid are involved in the pathophysiology of cerebral ischemia. In contrast to this, Jugdutt has shown that indomethacin increases the size of myocardial infarct mass in dogs.

The Hossmann model proposes that the formation of ischemic cerebral edema is due in part to the redistribution of ions down their concentration gradients, when ion homeostasis can no longer be maintained. Therefore, it may be expected that an agent which alters the rate of formation of edema may simultaneously affect the critical levels of blood flow at which K and Ca homeostasis is disturbed.

This study was designed to test the effect of indomethacin preloading upon the formation of ischemic cerebral edema and upon the critical blood flow thresholds for the disruption of K and Ca homeostasis.

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Stroke. 1982;13:11-17
doi: 10.1161/01.STR.13.1.11

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