Clinical Classification and Manifestations of Stroke

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Stroke is a focal neurologic deficit of acute onset of presumed vascular origin. This definition implies a clinical event, with a focal dysfunction of the central nervous system (CNS), which is likely to be secondary to a disease involving the vessels and circulation. Stroke can be ischemic or hemorrhagic.

Ischemic stroke is the most common type (85–90%) (1,2). It corresponds to transient or persisting interruption or decrease of blood flow in a focal area of the brain, usually in a partial or complete territory of a cerebral artery. The cause is usually an occlusion of the corresponding artery, but rather from embolism than from in situ disease, except in the very small branches which perforate the parenchyma. The lack of focal blood supply triggers a bioelectrical and metabolic cascade that ultimately results in tissue necrosis (cerebral infarct), if blood flow is not restored during the very first hours. If the occluded artery reopens early with subsequent improvement or normalization of blood supply, the tissue lesion will be small or absent and its clinical expression will be a transient ischemic attack (TIA). Transient ischemic attacks constitute about 10% of all strokes. About one third precede the development of a permanent stroke, and about one third of permanent strokes have been preceded by one or more TIAs. Most TIAs last only a few minutes (15 to 30 minutes as a rule) not hours. When the deficit lasts longer than 1 hour, the rate of underlying infarct may be as high as 75% (3). In this situation, the term TIA can be misleading, and cerebral infarct with transient signs (CITS) has been proposed (4). Other terms [prolonged TIA, reversible ischemic neurologic deficit (RIND), etc.] have been proposed in patients with deficits lasting more than 24 hours but less than 2 or 3 weeks, but these subdivisions may not be warranted in that prognosis of patients with TIA or cerebral infarct with minor deficit (minor stroke) is remarkably similar (5,6). However, it is important to specify the presence or absence of a visible infarct [on computerized tomography (CT) or magnetic resonance imaging (MRI)] in patients with a short-lasting event, because there is evidence that, if an infarct is present, a second stroke can be associated with more severe and long-lasting dysfunction (7).

Cerebral hemorrhage accounts for no more than 15% of strokes (1,2), except in
non-White populations. It is due to the irruption of blood into the brain parenchyma usually because of rupture of a small perforating artery, with disruption of the parenchyma, edema, and subsequent necrosis.

A hemorrhagic infarct corresponds to bleeding into an area of infarction. At necropsy, most infarcts show extravasation of blood and hemorrhagic zones, but in clinical practice, the term is restricted to visible hemorrhage on CT (or MRI). Sequential CT studies show that over one third of infarcts may show visible hemorrhage within 1 month of stroke onset (8). The appearance is typical, with heterogeneous and gyriform areas of bleeding within a well-demonstrated infarct. However, in some patients, the bleeding may take the form of a frank hematoma within the infarct, which is no longer visible (intra-infarct hematoma) (9), so that differentiation of a primary ischemic process with secondary bleeding from a primary cerebral hemorrhage can be exceedingly difficult. The two most widely used types of stroke classifications are (a) topographic; (b) etiologic. This is legitimate since recognition of one subtype can alter the patient management, and because there are significant links between the topography and the etiology of a stroke. Other useful categorizations also consider age (e.g., 0–15; 16–45; 46–75; > 75 years), or stroke features such as type of onset. Nonprogressive, progressive, and fluctuating are the three main types of onset which are usually considered.

CAUSES OF CEREBRAL INFARCTS

Most cerebral infarcts are linked to atherosclerosis in the cardiovascular system, though usually through an indirect mechanism (embolism). Early and sequential angiography has shown that distal intracerebral occlusions are present in most patients within the first 24 hours and disappear thereafter. Overall, the three leading causes of cerebral infarcts are:

(i) Extracranial large artery disease (40%), usually with artery-to-artery embolic occlusion of intracerebral arteries. The embolic material can be either fibrin-platelet aggregations or from a red thrombus which usually occludes the extracranial artery. Hemodynamic mechanisms may play a role in large artery occlusion or severe stenosis, but they are usually not prominent.

(ii) Cardioembolism is the presumed cause of infarct in at least 25% of infarcts, though definitive proof of embolization from the heart is usually lacking. The most common potential cardiac sources of embolism include: (a) dysrhythmias: atrial fibrillation, sick-sinus syndrome; (b) structural changes: rheumatic heart disease, acute myocardial infarct, left ventricular akinetic segment or aneurysm, and dilated cardiomyopathy (10). Hemodynamic phenomena associated with decreased cardiac output are an uncommon cause of stroke, but are also commonly overlooked as a potential contribution or aggravating factor (11).

(iii) Small artery disease (microangiopathy) accounts for about 15% of cerebral infarcts. It corresponds to in situ stenosis and occlusion of deep perforating branches
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of the hemispheres and brain stem. In contrast to pial arterial branches, these perforators lack significant anastomoses so that collateral supply is not available when occlusion develops, so occlusion leads to a small infarct (≤ 15 mm in diameter) limited to the territory of the occluded perforator (lacunar infarct) (12). Hypertension is the main risk factor for small artery disease.

Between 5% and 25% of patients may have coexistence of these three leading causes and it may be difficult to determine the precise etiology in these patients. Other potential causes of cerebral infarct are numerous but less common (2). Many of these uncommon causes of cerebral infarct become important in younger patients (≤ 45 years). In this age group, the three most common determined causes of ischemic stroke are (13):

(i) **Cardioembolism.** Up to 40–50% of the patients may have a patent foramen ovale as compared to 15–20% in age- and sex-matched controls (14,15). Though paradoxical embolism is difficult to prove in most instances, it is often suspected on the basis of triggering factors with Valsava maneuver. The association with interatrial septal aneurysm suggest that a paradoxical embolism may occur without a venous source (16).

(ii) **Arterial dissection,** mainly in the carotid circulation (17), is classically divided into spontaneous and traumatic. Cerebral infarct develops when the arterial lumen is severely narrowed or occluded with the development of local thrombosis and subsequent embolization.

(iii) **Migraine stroke** remains controversial. It may be a specific entity but should be diagnosed only with strict criteria that underscore the temporal relationship between a prolonged attack of migraine with aura ending in cerebral infarction (18).

Changing patterns of the etiology of ischemic stroke in the young may appear in the near future, as suggested in recent studies emphasizing the role of inherited mitochondrial dysfunction and familial genetic cases (19).

The main similarity between stroke in the young and stroke in the elderly (>75 years) is the importance of cardioembolic sources (20), while large artery atheroma and small vessel disease are less prominent (probably because patients with these conditions will have died from vascular disease before the age of 75).

CEREBRAL INFARCTS: CLASSIFICATION BY VASCULAR TERRITORY

Middle Cerebral Artery Pial (Superficial) Territory Infarcts

The main characteristic of this territory is that it has an extensive collateral system, so that multiple embolism is usually necessary before infarction occurs. The cause of embolism is artery-to-artery in at least one third of the patients and cardiac in one quarter, the latter being especially common in infarcts in the territory of the lower division of the middle cerebral artery (21).
Anterior Cerebral Artery Territory Infarcts

These are at least 20 times less common than middle cerebral artery territory infarcts, but their etiologic patterns do not differ (22).

Subcortical Infarcts in the Anterior Circulation

Infarction limited to the territory of one single perforator has been called lacunar, and is usually associated with in situ small artery disease linked to chronic hypertension, often asymptomatic. Larger infarcts encompassing the territory of several perforators are usually symptomatic and are not associated with small vessel disease. Artery-to-artery embolism, cardioembolism, and intracranial large artery stenosis occluding the mouth of perforating branches are the most common etiologies. The best known of these larger subcortical infarcts are the striatocapsular infarcts (lenticulostriate branches off the middle cerebral artery trunk), the large infarcts in the anterior choroidal artery territory, and the large centrum ovale infarcts (white matter medullary branches).

Classification of the subtypes of cortical infarcts depends on size, potential etiology, and clinical manifestations. The term lacunar syndrome was initially introduced to report any possible manifestation of lacunar infarcts (23), but its commonly accepted meaning is syndromes that are suggestive of lacunar infarcts. Confusion between these two definitions has led to innumerable misleading assumptions on subcortical infarcts. Syndromes suggestive of lacunar infarction are few, and strict criteria of the distribution of motor or sensory deficits must be used (in particular, combined involvement of face, upper limbs, and lower limb in pure motor hemiparesis, pure sensory stroke, ataxic hemiparesis, and sensorimotor stroke) (24).

Superficial Posterior Cerebral Artery Territory Infarcts

As for the posterior middle cerebral artery infarcts, the frequency of embolic sources is high.

Thalamic Infarcts

These develop in four main arterial territories: paramedian (first segment of posterior cerebral artery), thalamogeniculate, posterior choroidal (second segment of posterior cerebral artery), and tuberothalamic (posterior communicating artery) (25).

Border Zone Infarcts

There are two main types of border zone infarcts: (i) watershed infarcts develop at the level of the most distal anastomoses between two main branch territories; (ii)
other border zone infarcts occur in the area between the terminal territory of deep and superficial perforators (subcortical border zone infarcts). They are associated with ipsilateral severe carotid disease (26).

**Large Hemispheric Infarcts**

These combine the superficial and deep territory of middle cerebral or posterior cerebral arteries, or correspond to middle cerebral artery with posterior or anterior cerebral artery territory involvement.

**Brain Stem Infarcts**

Partial infarcts involve either the paramedian or the lateral territory. These infarcts are not uncommonly associated with severe local disease of the basilar artery or distal vertebral artery, which may occlude the origin of perforating or short circumferential branches.

**Cerebellar Infarcts**

Posterior inferior cerebellar artery territory infarcts are often associated with atheromatous disease of the vertebral artery, whereas superior cerebellar artery territory infarcts are associated with cardioembolic sources. Anterior inferior cerebellar artery territory infarcts are uncommon (27).

**CLINICAL MANIFESTATIONS OF BRAIN INFARCT**

The diagnosis of cerebral infarction is supported by the presence of previous transient ischemic attacks, associated cardiovascular disease and a neurologic deficit which is immediately or rapidly stabilized after onset. Large artery disease may be present if there is a history of previous transient ischemic attacks or transient monocular blindness in the same territory and an immediate complete neurological deficit. Cardiac embolism is suspect with a history of previous transient ischemic attacks or infarcts in different arterial territories, abrupt onset, loss of consciousness at the onset, and an isolated branch artery syndrome. Lacunar infarct should be evoked in an elderly patient with longstanding hypertension with proportional faciobrachiocrural hemiparesis, hemihypesthesia, or both, as well as in the presence of dysarthria-clumsy-hand syndrome and ataxic hemiparesis (lacunar syndromes) without cognitive disturbances (except in the case of thalamic infarcts).

However, none of these criteria is absolute and, for instance, 8% of patients with cerebral hemorrhage report previous transient ischemic attacks. Also, small deep hemorrhages sometimes lead to pure motor hemiplegia or other lacunar syndromes. In clinical practice, we have often found that brain hemorrhages should be suspected
when the neurological picture does not agree with the well-established vascular syndromes, suggesting the underlying process may trespass on the boundaries of the usual arterial territories.

Vital Functions

High blood pressure with a spontaneous decline pattern within 2 weeks following stroke are found in patients with acute ischemic or hemorrhagic lesions. Hemispheric strokes can produce contralateral reduced chest wall movements, reduced diaphragmatic excursion, or Cheyne-Stokes rhythm. Central hypoventilation involving automatic and voluntary respiration may occur in lesions involving the nucleus ambiguus and the nucleus tractus solitarius. Loss of automatic respiration (Ondine's syndrome) and obstructive and mixed sleep apnea syndromes are observed in lateral medullary infarcts (28). Secondary cardiac dysrhythmia, including transient atrial fibrillation, is found in over 5% of patients during the first hours after a stroke. It is found mainly with parieto-insular and caudal brain stem lesions involving nuclei with arrhythmogenic capacity (29). Fever is rarely due to stroke. More commonly, it is an indicator of intercurrent infection. Noninfectious excessive sweating is found in ischemic or hemorrhagic brain stem lesions. Transient unilateral hyperhidrosis limited to the contralateral upper face, shoulder, or arm may occur in large infarcts or in the middle cerebral artery territory and in small infarcts of the frontal operculum (30). Ipsilateral hypohidrosis may occur in pontine and medullary infarction (31). Focal areas of decreased skin temperature on the hemiparetic side are found in 5% of patients with large cortical, capsular, and pontine infarcts (32).

Higher Cerebral Functions

Cerebral strokes may lead to many neuropsychological and behavioral disturbances. Several of these have been predominantly associated with left-sided lesions, but this probably often reflects a bias due to language abnormalities associated with left-sided brain damage. Although the frontal lobe is affected in a large proportion of strokes, paradoxically the behavioral disturbances associated with frontal stroke have been rarely studied (33). A similar situation occurs with right temporal or occipital lobe dysfunction. These may, therefore, be overlooked during clinical examination because they are poorly recognized.

Broca's aphasia occurs with damage to the territory of cortical branches of the middle cerebral artery involving the inferior frontal areas including the underlying white matter. However, more restricted lesions in the frontal operculum (Broca's area) produce only a transient deficit with delayed initiation of a grammatically correct language. Transcortical motor aphasia with proximal limb paresis is observed in lesions of the territory of the left central artery involving the frontal operculum and the underlying white matter. Conduction aphasia is usually associated with bucco-lingual apraxia, a slight right-sided facial weakness and right hemisensory
disturbances in relation to an infarct in the territory of the anterior parietal artery. 
Wernicke’s aphasia is typically due to infarct in the territory of the posterior branches 
of the middle cerebral artery.

Transcortical sensory aphasia is found in lesions immediately posterior to Wernicke’s area. Persisting global aphasia occurs in large perisylvian infarcts or extensive lesions of the basal ganglia and overlying insula. It can also develop without hemiparesis in double separate temporal and frontal strokes sparing the central region. Global aphasia is the most common aphasia type in the acute phase of stroke, but it evolves over a few hours to a few days toward more specific aphasia types according to stroke topography.

Lesions in the frontal lobe, parietal lobe, and thalamus may produce hemineglect. Hemineglect occurs more commonly and is more severe with right hemisphere lesions. In this case, it is often accompanied by denial of illness, dressing apraxia, constructional apraxia, and motor impersistence.

Visual Fields

Visual field loss caused by stroke may be detected or at least suspected during the initial clinical examination. The optic tract and the temporal optic radiations (Meyer’s loop) are involved in lesions of the choroidal anterior artery. The parietal lobe and the occipital cortex are involved in lesions of the middle cerebral artery and posterior cerebral artery, respectively.

Pupils

Asymmetric pupils may be due to oculomotor nerve or sympathetic involvement. Unilateral miosis with Horner’s syndrome may be found in extensive middle cerebral artery infarction and in hypothalamic and brain stem infarctions. Bilateral miosis with preservation of the light reflex is observed in pontine lesions. Bilateral mydriasis is observed in mesencephalic infarction.

Eye Movements

Conjugate eye deviation toward the lesion is found in approximately 20% of patients with cerebral hemispherial stroke, especially on the right side. Contralateral eye deviation (“wrong way eye”) is nearly specific for hemorrhage. In infratentorial lesions, a contralateral conjugate eye deviation (the patient looks at his/her hemiplegia) may be found in unilateral lesions of the pontine paramedian reticular formation, but in general, conjugate eye deviation is less common than in the supratentorial lesions. Slowness or absence of vertical saccadic movements is found in patients with lesions involving the rostral interstitial nucleus of the medial longitudinal fasciculus.
Complex and dysconjugate eye movement disorders may occur in stroke and cannot be detailed here.

**Motor Dysfunction**

Hemiparesis or hemiplegia with different degree of severity are common in patients with stroke. Faciobrachiorcular hemiplegia is found with large or deep middle cerebral artery infarcts, but can also be due to pontine or anterior choroidal artery territory infarction. When isolated, it is highly suggestive of lacunar choroidal artery involvement of the internal capsule or pons. Hemiparesis with crural predominance is found in anterior cerebral artery territory infarction and anterior junctional infarcts, usually associated with transcortical motor aphasia in left-sided lesions. Rarely, hemiparesis with a facio-brachial predominance may also be found in anterior cerebral artery infarction. A predominantly proximal paresis of the arm with a greater difficulty in elevation and abduction may be observed with lesions limited to the premotor cortex in the middle cerebral artery territory or the supplementary motor area in the anterior cerebral artery territory (34). Infarction in the territory of the cortical branches of the middle cerebral artery usually leads to hemiparesis with facio-brachial predominance. An unusual pattern of motor deficit, with bilateral proximal brachial paresis ("man-in-the barrel"), may be seen in bilateral anterior junctional infarcts involving the precentral gyri. On the other hand, when junctional infarct is mainly subcortical in the white matter, a pattern of cerebral paraparesis may occur.

A small white matter (centrum ovale) infarct may cause hemiparesis in any possible combination including isolated upper limb monoparesis.

Unilateral lower cranial nerve palsy of supranuclear origin is found in corona radiata and capsular genu infarction (7). Bilateral lower cranial nerve palsy with marked dysarthria and facial-pharyngeal-lingual palsy sparing the emotional movements (automatic-voluntary dissociation) is found in bilateral frontal operculum or immediate deeper lesions.

Hemiparesis is uncommon in posterior cerebral artery infarctions, but it may occur when its midbrain territory is involved.

**Sensory Abnormalities**

Hemihypaesthesia with crural predominance and rarely with facio-brachial predominance is found in anterior cerebral artery territory infarction. Hemihypaesthesia with facio-brachial predominance usually associated with hemiparesis is characteristic of middle cerebral artery territory infarction. However, a pseudothalamic pattern with predominantly elementary sensory loss in the hemibody, including the trunk and reaching the midline, may be found with lesion of the infero-anterior portion of the parietal lobe (35). Sensory loss with a pseudospinal or pseudoradicular pattern is rarely found in patients with parietal stroke.

Pure sensory loss is not specific for a particular topographic pattern of lesion,
although it is most commonly seen with lateral thalamic infarction. Partial forms of pure sensory stroke may develop with lesions of the frontoparietal cortex, corona radiata, thalamus, midbrain, and pons.

Ataxia and Cerebellar Signs

Limb incoordination associated with trunk ataxia, gait ataxia, and speech disturbances may be a prominent finding in patients with cerebellar or brain stem stroke. However, ataxia restricted to the upper limb, associated or not with negative myoclonus (asterixis), may be observed in patients with parietal stroke; a cortical or pseudothalamic pattern of sensory loss may coexist but not always. Ataxia associated with motor deficit is found in patients with cortical frontal (anterior cerebral artery territory), centrum semiovale (middle cerebral territory), internal capsule, midbrain and pons infarction (ataxic hemiparesis). An interesting pattern of ipsilateral ataxia mainly restricted to the upper limb with crural paresis is typical of superficial anterior cerebral artery infarct in the paracentral area, although it has been previously attributed to deeper lesions (36).

Approximately one quarter of patients with a thalamic infarct have ataxia. Three patterns of ataxia related to thalamic lesions have been identified: (i) hypaesthetic ataxic hemiparesis, (ii) ataxic hemiparesis, and (iii) hemiataxic hypaesthesia. Variants of ataxic hemiparesis include dysarthria-clumsy-hand syndrome caused by contralateral pontine lesion and ataxic tetraparesis associated with pontine or bilateral capsular stroke.

Infarction limited to the territory of the lateral branch of the posterior inferior cerebellar artery is characterized by ipsilateral limb ataxia involving the arm and leg, without dysarthria, truncal ataxia or alteration in eye movements. Limb ataxia, trunk ataxia, nystagmus, and dysarthria are the prominent signs in infarct in the territory of the superior cerebellar artery. Rarely, isolated axial lateropulsion may be the only manifestation of an infarct involving the lateral branch of superior cerebellar artery.

Abnormal Movements

Abnormal movements may rarely be the isolated manifestation of stroke. Usually they are associated with motor or sensory deficit. Contralateral hemichorea, hemiballismus, or hemidystonia can be observed in cortical, junctional, and basal ganglia stroke. Focal dystonia, asterixis, myoclonus, and stereotyped motor behaviors can also occur.

Thalamic-astasia, characterized by an inability to stand or walk despite minimal weakness or marked sensory disturbance, may be found in thalamic stroke (37).
CEREBRAL HEMORRHAGE

Etiology

Chronic hypertension is the most common etiological factor in cerebral hemorrhage but its role is declining and only about 50% of cases are now attributed to it. Bleeding occurs at the level of small penetrating arteries, which may develop microaneurysmal changes (Charcot-Bouchard’s aneurysms). Acute hypertension causes cerebral hemorrhage not only in the setting of chronic hypertension but also independently, in association with alcohol or drug abuse, exposure to extreme cold, trigeminal nerve stimulation, after carotid endarterectomy, and after heart surgery (38). The proportion of nonhypertensive causes of cerebral hemorrhage is variable and depends on age and site of bleeding, exceeding 50% in patients below 40 years. Between 1 in 10 and 1 in 5 cases of cerebral hemorrhage are due to cerebral amyloid angiopathy, a sporadic condition characterized by deposits of amyloid in the media and adventitia of small- and medium-sized arteries. This diagnosis should be assumed in nonhypertensive patients over 60 years with recurrent subcortical lobar hemorrhages with or without Alzheimer’s type of dementia. Other causes include anticoagulation thrombolysis, congenital or acquired coagulopathies, vascular malformations, arteritis associated with drug abuse, cerebral venous thrombosis, trauma, and aneurysms.

Clinical Presentation

Transient ischemic attacks may precede cerebral hemorrhage in 5–10% of patients. Two out of 3 cases of hemorrhage stabilize rapidly, while one 1 of 3 has a progressive course over hours or even days (39). Cerebral hemorrhage usually occurs in the daytime during physical activity; the clinical picture classically includes headache and vomiting (40–50% of cases), decreased alertness (60%), systolic hypertension (90%), and meningeal signs. On the other hand, up to 10% of cases may produce a lacunar syndrome (2). Cerebral hemorrhage can also mimic ischemic stroke with short lasting neurologic symptoms without headache or clouding of consciousness. It can also occur in a pseudotumoral form with seizures and a focal deficit progressing over weeks. Overall, seizures are not common (10%) in cerebral hemorrhage, but in stroke, a seizure at onset may suggest a cerebral hemorrhage. Multiple hemorrhages are found in 2% of cases, often associated with hypertension. Intraventricular bleeding may present with a subarachnoid-bleeding-like picture (headache, meningism, confusion). Bleeding into the ventricles occurs in one third of cerebral hemorrhage cases, but primary intraventricular bleeding is rare (Moya-Moya disease, angiomas, anticoagulation, and bilateral carotid disease).
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Deep Supratentorial Cerebral Hemorrhage

About 50% of cerebral hemorrhage cases involve deep supratentorial structures and among these most (>70%) are due to chronic hypertension. Putaminal hemorrhages are the most common form. They present with hemiparesis, decreased alertness, and ipsilateral conjugate eye deviation. Smaller lesions can present with lacunar syndromes or paroxysmal movement disorders. Thalamic hemorrhages should be suspected in patients with hemisensory (-motor) dysfunction, vertical eye movement disturbances, bilateral miosis, and behavioral abnormalities following decreased alertness. Hemorrhages of the head of the caudate nucleus are characterized by a subarachnoid-bleeding-like picture or by a slight hemiparesis with prominent behavioral changes.

Lobar Cerebral Hemorrhage

One third to over one half of cerebral hemorrhage cases involve superficial (lobar) structures. A nonhypertensive origin is common (50–70% of cases). Headache, vomiting, meningism, and seizures are more frequent than in deep hemorrhage, but decreased alertness, severe hemiparesis and death are less frequent.

Brain Stem Cerebral Hemorrhage

The most common form involves the basis of the pons. It is usually of hypertensive origin, prognosis is commonly poor, with coma, pinpoint pupils, tetraparesis, and eye movement disorders.

Cerebellar Cerebral Hemorrhage

Bleeding usually develops in the vicinity of the dentate nucleus and is often due to chronic hypertension. It presents with headache, vomiting, dizziness, and inability to walk. Gait and limb ataxia, dysarthria, gaze palsy, and clouding of consciousness are common. The clinical course is often unpredictable, but prognosis is poor if coma develops.

REFERENCES


**DISCUSSION**

*Dr. Guesry:* Do you have any information on the frequency of clinical manifestation which could induce nutritional problems either during the acute phase or during the recovery period, for example swallowing defects, diminution of consciousness, coma, or motor problems?

*Dr. Bogousslavsky:* There are two phases. The first phase, with swallowing difficulties, is present in nearly every patient with acute stroke. The incidence of bronchial aspiration is probably close to 100% in the acute phase of stroke because of swallowing disturbances. The second phase, with persistent swallowing disturbance inducing nutritional problems, is much rarer. It may be linked to two aspects: either neuropsychological, that is, disturbances of higher cortical functions such as frontal lobe dysfunction (some patients have bulimia or do not do anything to feed themselves), or second, to direct involvement of neural commands, as in patients with bilateral facial masticatory palsy. These problems are much less common, certainly less than 5–10% and probably less than that if we look at the patients after a few months.

*Dr. Homberg:* I was interested in the converse of the neglect syndrome that you mentioned, with patients having parietal lesions somewhat anterior to the classical neglect areas. Do you have evidence of any further attentional problems in these patients, not just related to proprioceptive problems but also to visual extraceptive problems?

*Dr. Bogousslavsky:* Within the limits of the test, which has to be done within the first few days because the syndrome is transient, we have no evidence of any other attentional disturbances, there is no over-interest in the visual field or neglect of other aspects—if you perform the classical tests, they are normal. The syndrome is probably related to the feeling of strangeness associated with the acute sensory disturbance; the patient feels that the left side of the body is really odd.

*Dr. Hossmann:* Did you find any association between the topography of ischemic lesions and memory deficits in your patients?

*Dr. Bogousslavsky:* When we address memory as a single problem, we rarely see specific memory problems in single strokes. Perhaps it is more evident if you have repeated strokes, but in single strokes, the purest memory disturbances that we have seen was in thalamic infarcts involving the anterior part of the thalamus, where memory disturbances can be present on their own without other neuropsychological or neurologic disturbances. Otherwise, this is rather uncommon for a single stroke.

*Dr. Stähelin:* Is there an age-dependence effect on the memory dysfunction so that elderly persons with a single stroke have more memory dysfunction than younger ones?

*Dr. Bogousslavsky:* I think it is true that neuropsychological dysfunction can be more prevalent after a single stroke in elderly persons because recovery is less; however, the features need to be matched—for instance, the size and location must be similar because strokes may differ in younger individuals.

*Dr. Hennerici:* This is an interesting feature. The final topography of the lesion is different
from what the clinician sees if he sees the patient very early at a time when most imaging scans don’t show anything and where the picture is probably not related to the final lesion but to the ischemic area, which is far larger than the core. Thus in combination, in an older subject with a repeated history of strokes, we have a much more complicated situation, as there are definite fixed lesions from previous strokes and a mixture of an extending acute lesion and a shrinking one after a few days, down to the final lesion. I wonder if we have really studied this carefully enough under clinical conditions. Quite a few of the concepts which you addressed as misconcepts come about because of these different conditions in the patient. In particular, cases with multiple strokes have not been well studied.

Dr. Wolf: I wonder if we are really talking about cognitive dysfunction rather than memory. For neurologists, memory is somewhere in the hippocampus and median temporal lobe and not a global loss of cognitive function.

Dr. Bogousslavsky: I think this is always a matter of terminology. For instance, the term dementia has been used for many years and is now common parlance, but in a way, the term does not apply to what we see in stroke patients; even in patients with multiple strokes, it does not fit what is behind the concept of dementia and the term that was proposed by Vladimir Hachinski of vascular cognitive impairment may be more accurate if more cumbersome.

Dr. Feller: As a gastroenterologist, it seems to me that many patients with stroke are getting into big nutritional difficulties, though this may be because my neurological colleagues call me only for these patients. Besides dysphagia problems, do you think that stroke affects appetite in some patients? Even in patients with no depression, I have the feeling that some of them just stop eating or have a different eating pattern than before the stroke, whereas some eat in excess.

Dr. Bogousslavsky: The problem is somewhat complicated by the emotional disturbances which are secondary to stroke. We are studying the problem of emotion immediately after stroke and, of course, appetite, as you know, is an item in the Hamilton depression scale, so we cannot just look at appetite in the absence of all the other patterns. But it would be interesting to look at specific disturbances such as appetite loss or appetite change in the absence of marked depressive features. To my knowledge, this has not been done yet.

Dr. Ginsberg: Do you think there may be a central cause of dysgeusia, that is, abnormality of taste perception that is not of peripheral but of central origin? If this were possible in general, it might occasionally be possible in stroke.

Dr. Bogousslavsky: I don’t think we have seen a patient with a specific dysgeusia; probably it can occur because it is seen in epilepsy and other diseases, but I don’t remember seeing a patient.

Dr. Feller: The dysgeusia question is very interesting. We have done a study in institutionalized elderly people and there is a very high prevalence of zinc deficiency, which is known to induce dysgeusia.