Section 1 | **Clinical manifestations**

Chapter

1

Hemiparesis and other types of motor weakness

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Motor weakness concepts

Motor weakness has been defined as a difficulty in generating the necessary voluntary muscle force for effective motor and movement performance. Movement (to be in motion) means spatial moving of any part of the body. It is produced by muscle contraction, usually of striated muscle, and may be voluntary, automatic (involuntary), or reflex, as a result of muscle strength involving the pyramidal tract, the motor nerves, the muscles, and the joints. For a movement to be correct, it is necessary that it involve functions of the many parts of the nervous system, specifically the senses, sensory nerves, cerebellum, and extrapyramidal system, to provide adequate muscle tone, coordination, and equilibrium.

Disturbances of voluntary movement in the form of paresis or paralysis are the usual consequence of pyramidal tract dysfunction, cerebrovascular disease being one of the causes. Voluntary movement disorders may also be due to non-pyramidal causes, such as loss of sensory or afferent pathways, coordination disorders related to cerebellar lesions, lesions of the extrapyramidal tracts, and alteration of cortical motor programming, causing apraxia or lack of initiative [1].

According to the topography of the lesion, movement dissociation may occur, which is a selective disorder of one of the three forms of movement (voluntary, automatic, reflex). In voluntary-automatic movement dissociation (e.g., cortical lesions), voluntary movements are abolished and automatic or reflex movements are preserved, whereas in automatic-voluntary movement dissociation (e.g., deep temporal lesions, lesions of the basal ganglia), automatic movements are abolished and voluntary movements are preserved [2].

The medical history is the initial and most important element to determine the characteristics of the motor deficit. Prior diseases of the patient, such as an embolism because of cardiac disease, risk factors like diabetes, and precipitating factors of the motor deficit such as a cervical trauma, are crucial features in the analysis of a motor deficit. Once motor weakness has developed acutely or subacutely, the chronology of the clinical course (continuous, intermittent, progressive), the distribution of the paralysis, and the accompanying symptoms may allow the establishment of the etiology [1,3].

The neurological examination may confirm the type of motor deficit and its accompanying manifestations, so that it is possible to suspect the topography, nature, cause, and mechanisms of production of the lesion. Once the diagnosis has been made, it is necessary to establish a differential diagnosis considering other etiological possibilities for the lesion. Subsequently, the results of complementary examinations will confirm or exclude the clinical diagnosis, and a therapeutic plan can be established [1,3].

Neuroanatomical considerations

In humans, approximately 60% of the corticospinal axons arise from the primary motor cortex, and the remainder from the premotor area, supplementary motor area, and the parietal lobe. The primary motor cortex contains a somatotopic representation of body parts (homunculus or manikin). However, recent investigations support the notion of multiple representations of different body parts within the primary motor cortex [4].

The corticospinal tract descends from the primary and more anterior supplementary motor cortex, converges within the corona radiata, and passes downward through the internal capsule (Figure 1.1). The positioning of the corticospinal tract in the internal capsule has a classical hypothesis suggested by Charcot in 1883 and documented by Déjérine in 1901 that localizes the corticobulbar or geniculate tract in the genu of the internal capsule and the corticospinal tract occupying the anterior portion of the posterior limb. This topographic hypothesis was still assumed by Testut, Bricort, and Lazhortes [4]. This traditional view suggested that fibers relating to the head pass through the anterior limb; those relating to the mouth, larynx, and pharynx are in the genu; those relating to the arm are in the anterior part of the posterior limb; while those relating to the leg lie more posteriorly. In contrast, Pierre Marie in 1902, and before that Bennet and Campbell in 1855, indicated a posterior localization of the pyramidal tract in the
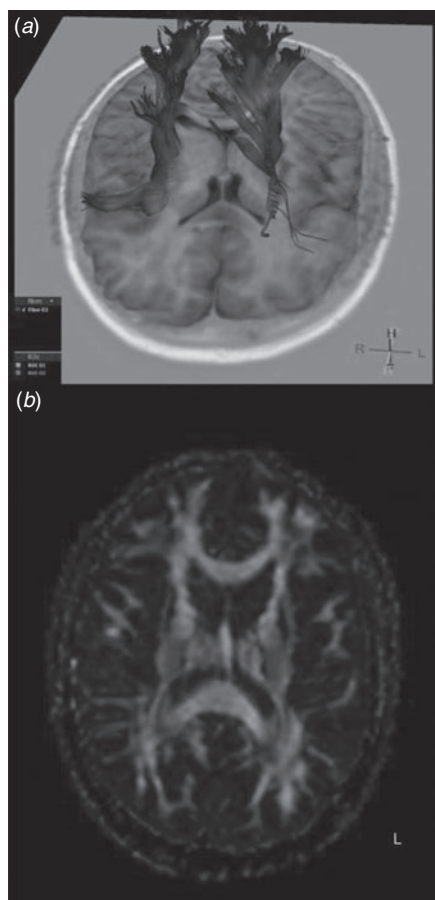
Figure 1.1. Diffusion tensor imaging acquired using a 3-Tesla magnet. (a): a 2D representation of the main brain bundles coded in color according to regional diffusion anisotropy. (b): a 3D display emphasizing pathways showing a superior-inferior direction, including internal capsule fibers and the pyramidal tract. (Courtesy of Dr. J. Pujol.) (See plate section for color version.)
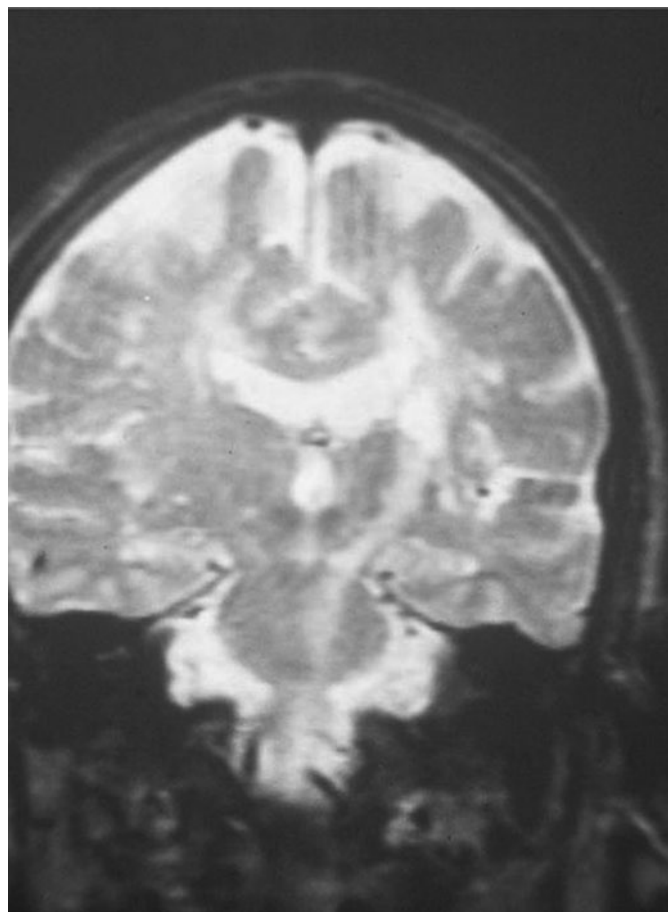
Figure 1.2. Coronal T2-weighted high-field (1.5-T) MRI demonstrating a long band of increased signal extending from a chronic capsular infarct to the medulla, suggestive of Wallerian degeneration of the pyramidal tract. (Courtesy of Dr. J. Pujol.) (See plate section for color version.)

internal capsule; however, these suggestions were not accepted by many academics [5].

Further anatomoclinical studies in patients with amyotrophic lateral sclerosis found the pyramidal tract in a more posterior position. These findings were also consistent with results of stereotactic stimulation studies carried out by Bertrand in 1963 and anatomoclinical observations of Eglander in 1975 and Hanaway in 1977 in patients with lacunar infarction, suggesting a more posterior location of the pyramidal tract (between the middle and posterior third of the posterior limb of the internal capsule), refuting the classical hypothesis. Finally, Rodd in 1980, in a detailed anatomical study, showed the characteristic rostrocaudal orientation of the pyramidal tract rather than a vertical and fixed orientation. The tract progressively shifted into the posterior half of the posterior limb; the fibers follow an oblique course through the capsule, becoming more posteriorly placed in the caudal (inferior) segments of the capsule [6]. Accordingly, the anteroposterior face–arm–leg somatotopic organization joins classical theory and more recent observations, making all of them valid. Tredici et al., in 1982, emphasized the possibility of individual anatomical variations in the location and distribution of the pyramidal tract, citing a clinically silent metastasis in the posterior limb of the internal capsule described by Rottenberg in 1976 [7].

The fibers then pass into the brainstem: cerebral peduncles of the midbrain and base of the pons, before entering the medullary pyramids (Figure 1.2). The facial nerve nucleus in the pons has a rostral portion from which fibers innervate the muscles of the upper face, while the motor caudal portion of the nucleus supplies fibers to the muscles of the lower face. The caudal loop of the fibers of the facial nerve descends as far as the medulla oblongata and explains why lesions of the medullary pyramid or medial medulla oblongata can be associated with contralateral upper motor neuron-type facial weakness.

At the junction of the medulla oblongata and spinal cord, some 75%–90% of the corticospinal fibers cross the midline and come to lie in an anterolateral position in the spinal cord, although a variable proportion remain uncrossed. These uncrossed fibers project to motor neurons in the medial part of the ventral horns, subserving axial and proximal muscles, corresponding with movements of the trunk [1,3].

The possibility of diaschisis should be considered, that is, disturbance or loss of function in one part of the brain due to a localized injury in another part. The term diaschisis was introduced by Von Monakow to describe the inhibitory effect

2

of a lesion in an area of the brain on other remote areas that are neuronally connected to it [8].

Clinical, topographical, and prognostic analysis of pyramidal paresis

The main cause of motor weakness is damage to the primary crossed corticospinal tract [3,4].

Assessment of the topographic distribution of the motor deficit is valuable to establish a clinicotopographic correlation [9]. The severity is helpful in the acute phase for determining prognosis, the potential risks and benefits of interventions (such as thrombolysis), and the functional management and rehabilitation of the patient. There are several methods of quantifying the severity of motor weakness, such as the Medical Research Council (MRC) scale, National Institute of Health Stroke Scale (NIHSS), and Scandinavian Neurological Stroke Scale, which have an operational definition of the grades of weakness, and moderately good inter-observer reliability [10].

Hemiparesis

Most patients with stroke (80%–90%) have motor symptoms or signs. A severe deficit, however, may be due to motor neglect, apraxia, or visuomotor ataxia and not weakness, and profound ataxia of gait may be associated with no motor deficit at all. Hemiparesis with uniform weakness of the hand, foot, shoulder, and hip is the most frequent motor-deficit profile (at least two-thirds of cases) [3,4].

Faciobrachiocrural hemiparesis

Hemiparesis with uniform weakness of the arm and leg associated with hemisensory deficit and speech deficit (dysphasia or dysarthria) usually indicates a large supratentorial lesion that involves the middle cerebral artery (MCA). Such patients have more severe weakness than do those with isolated hemiparesis. Distal predominance of the hemiparesis is usually related to cortical involvement, and speech abnormalities are caused by cortical lesions in the dominant hemisphere [3,4]. The syndrome of hemiplegia caused by an infarct of the entire surface territory of the MCA (e.g., Rolandic artery involvement) is characterized by brachial predominance of hemiplegia, not rarely involving mainly distal movements [9]. Hemiparesis of proximal muscles is due to a lesion involving the premotor cortex (e.g., pre-Rolandic artery involvement), but not the primary motor cortex. The paresis affects mainly those shoulder muscles that abduct and elevate the arm and all hip muscles to similar extents, the arm being functionally more affected than the leg [3,9]. The associated lesions are border-zone infarcts between the anterior and the middle cerebral arteries. In a clinical study of 34 hemiparetic patients after the first subcortical stroke, lesions in the proximal paresis group (n = 15) uniformly encompassed the middle part of the corona radiata, usually sparing the posterior half of the internal capsule [11].
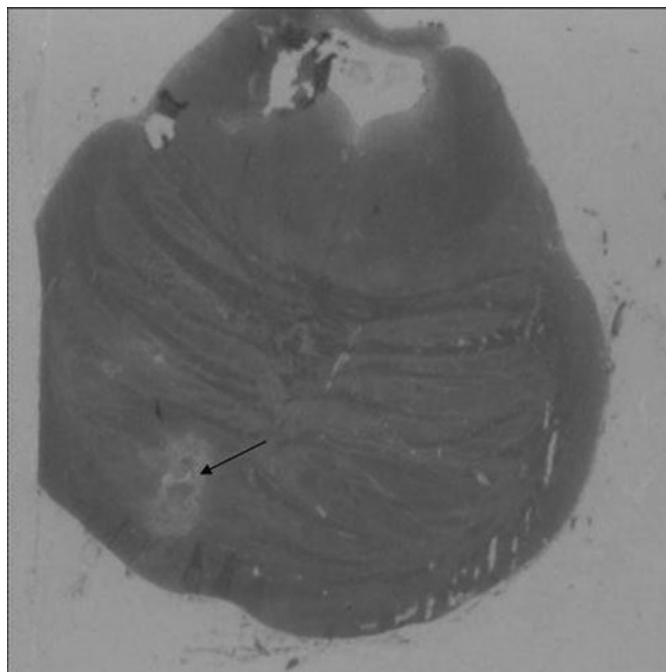
Figure 1.3. Anatomical specimen showing a lacunar infarct in the protuberance (arrow) in the distribution territory of a pontine paramedial arteriole (hematoxylin and eosin). (See plate section for color version.)

Prefrontal artery and anterior parietal involvement in non-dominant hemisphere lesions cause motor hemineglect. Large middle cerebral artery infarcts are usually caused by cardioembolism, internal carotid artery occlusion, or internal carotid artery dissection [4,9].

Pure motor hemiparesis

Pure motor hemiparesis, also known as pure motor stroke, is the most common of any lacunar form (between half and two-thirds of cases, depending on the series) [12–17]. In an acute stroke registry, pure motor stroke accounted for 12.7% of all first-ever stroke patients and for 50% of all lacunar syndromes [15]. The posterior limb of the internal capsule, corona radiata, and pons (Figure 1.3) are the most frequent topographies [12–16]. Infarcts in the mesencephalon (Figure 1.4) or medullary pyramid [18] especially have been reported.

Pure motor hemiparesis was the first clinically recognized lacunar syndrome. Clinical features include hemiplegia involving the face, arm, and leg, or incomplete hemiplegia involving the face and arm, or the arm and leg (brachiofacial or brachiocrural) proportionally or nonproportionally, in the absence of sensory deficit, visual deficit, and altered consciousness and impairment of higher brain functions. Only deficits involving the whole of the arm and face (brachiofacial), or the whole of the arm and leg (brachiocrural) should be accepted as partial lacunar syndromes, not more restricted deficits (e.g., hand only) that are more likely to be of cortical origin (Table 1.1) [19]. Lacunar infarcts located in the more posterior aspect of the

Table 1.1. Distribution of motor weakness in acute lacunar stroke patients with pure motor stroke and sensorimotor stroke included in the Sagrat Cor Hospital Stroke Registry [20]

Motor weakness	Pure motor stroke (n = 128)	Sensorimotor stroke (n = 41)
Face, upper limb, lower limb	112 (76)	39 (95)
Face, upper limb	6 (4)	1 (2.5)
Upper limb, lower limb	16 (10)	1 (2.5)
Face	6 (4)	
Upper limb	4 (3)	
Lower limb	4 (3)	

Percentages per column in parenthesis.
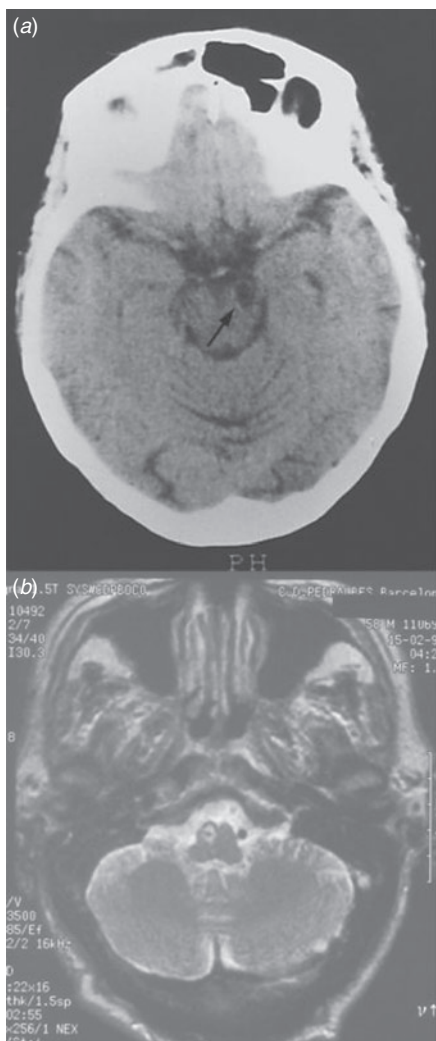
Figure 1.4. Lacunar infarction in the base of the mesencephalus (arrow) visualized by brain CT (a) and in the bulbar pyramid visualized by MRI (b), causing pure motor hemiparesis in both cases.

posterior limb of the internal capsule produce a predominantly crural motor deficit. Pure motor hemiparesis not due to lacunar infarction is found in 2%–15% of cases [20]. After Fisher and Curry's 1965 report of pure motor hemiplegia of vascular origin, several articles appeared challenging the lacunar origin by detailing a similar syndrome due to a variety of other causes, including nocardial abscess of the motor cortex, ischemia–edema after craniotomy for postoperative bleeding, internal carotid artery occlusion in the neck, and cerebral cortical surface infarction or ventromedial pontine infarction due to a propagating thrombosis of the basilar branch. A few such cases have even been reported after small brain hemorrhages (Figure 1.2) [20].

In a series of 222 consecutive patients with pure motor stroke, lacunar infarcts were found in 185 (85%) patients, whereas ischemic lacunar syndromes not due to lacunar infarcts occurred in 23 (10.5%) patients (atherothrombotic stroke in 12, cardioembolic stroke in seven, infarction of undetermined origin in three, and infarction of unusual etiology in one), and hemorrhagic lacunar syndromes in 10 (4.5%) patients [15].

Brachiofacial hemiparesis

Brachiofacial paresis is a stroke syndrome without involvement of the lower limb. In the majority of patients, faciobrachial hemiparesis is due to a cortical infarct in the superficial territory of the MCA. It is often seen in lesions involving the complete territory of the lenticulostrate arteries (subcortical hemispheric infarcts) or in the territory of the lateral lenticulostrate arteries. Large artery disease and cardioembolism are the main causes, while small vessel disease is infrequent. In a recent study, four of 22 patients with a brachiofacial pure motor stroke had a non-lacunar cortical infarct in the territory of the superficial MCA [21]. Pure motor hemiparesis of brachiofacial distribution due to a lacunar infarct is found in only 4% of patients with pure motor lacunar syndromes (Table 1.1) [22].

Brachiocrural hemiparesis

Sparing of the face in a pure motor stroke raises suspicion of a lower brainstem lesion rather than a supratentorial lesion.

Isolated monoparesis (brachial or crural) is rare (1.2%–2.5% of all strokes). The majority of such patients have brachial monoparesis; crural monoparesis is present only in 0.2% of all strokes. Pure motor monoparesis is almost never due to a lacunar infarct [19,23]. Isolated monoparesis can be the clinical presentation in up to 4%–6% of patients with lacunar infarcts. Monoplegia is usually associated with small infarcts in the cerebral cortex and adjacent subcortex in the territory of the MCA. Isolated monoparesis, therefore, is not a lacunar syndrome. In a clinical series of 52 patients with isolated monoparesis, cardioembolism was the cause in 15.7% of patients, atherosclerosis in 9.8%, small artery disease in 39.2%, and hemorrhagic stroke in 23.5% [24,25]. In a recent clinical study, pure monoparesis of the leg was related to infarctions located in the posterior limb of the internal capsule, corona radiata, and the anterior cerebral artery (ACA) territory [26].

4

Dysarthria–clumsy hand syndrome is a rare lacunar syndrome with an excellent prognosis. In a series of 2500 acute stroke patients included in a hospital-based prospective stroke registry over a 12-year period, 35 patients were identified as having dysarthria–clumsy hand syndrome. Dysarthria–clumsy hand syndrome accounted for 1.6% of all acute stroke patients, 1.9% of acute ischemic stroke cases, and 6.1% of lacunar syndromes [27]. Clinical manifestations include moderate or severe dysarthria with central facial weakness, homolateral hyperreflexia with Babinski's sign, and weakness of the hand with impairment of tasks requiring manual ability (e.g., writing), without an important associated motor deficit. Some authors consider dysarthria–clumsy hand syndrome as a variant of ataxic hemiparesis [19]. Infarcts are usually found in the internal capsule (in the anterior limb, genu, or near the genu) and the pons (in the paramedian rostral sites), although other topographies, such as the cerebellar peduncules and the corona radiata, have been described [28–30]. The absence of neurological disability in 46% of patients indicates that dysarthria–clumsy hand is the classical lacunar syndrome with the most favorable outcome [27]. Dysarthria–clumsy hand syndrome not due to lacunar stroke is found in less than 7% of cases, and may be caused by non-lacunar infarcts, cerebral hemorrhage, or infection.

Isolated facial paresis is a rare manifestation of stroke. Isolated facial paresis can be the clinical presentation in up to 6% of patients with lacunar infarction. In a clinical series of 227 patients with lacunar infarcts, neuroimaging studies revealed lacunar infarct in the genu of the internal capsule in three patients and in the pons in one [31]. Another study emphasized a capsular-corona radiata localization. These patients usually also had dysarthria. Isolated dysarthria or isolated facial paresis can be considered as an extreme continuum of dysathria–facial paresis syndrome, usually associated with lacunar infarcts in the corona radiata, basal ganglia/internal capsule, or pons [32].

The brain motor area of the hand is localized in a specific segment of the precentral gyrus as an inverted omega or epsilon in the axial plane. Lesions at this location may cause isolated hand palsy. Lesions of the parietal lobe may also cause isolated hand palsy [33]. Selective weakness of a particular group of fingers due to cortical infarcts in the precentral gyrus ("peripheral pseudoparalysis") has been described. Traditionally, a discrete somatotopic arrangement for individual fingers, with the radial fingers represented laterally and the ulnar fingers medially, has been assumed. Two possible etiological patterns according to motor involvement have been suggested, including infarcts associated with carotid atherothrombosis for ulnar fingers and cardioembolic infarction for radial fingers [4,33]. Predominant weakness of the index finger due to contralateral ischemic stroke in the precentral region has been reported [34].

Bilateral weakness may be caused by spinal cord, bilateral cerebral hemispheric, or brainstem infarction. Paralysis of extremities and the lower cranial musculature with sparing of

Table 1.2. Variables independently associated with lacunar syndrome not due to lacunar infarct [20]

Variable	Odds ratio (95% confidence interval)	P
Model based on demographics, vascular risk factors, and clinical variables		
Atrial fibrillation	4.62 (2.56–8.36)	0.0001
Sensorimotor stroke	4.05 (2.28–7.19)	0.0001
Limb weakness	2.09 (1.03–4.26)	0.042
Sudden onset	2.06 (1.25–3.37)	0.004
Age	0.96 (0.94–0.98)	0.001

consciousness (the locked-in syndrome) results from bilateral corticobulbar and corticospinal tract lesions, usually caused by basilar artery occlusion or pontine hemorrhage. Bilateral anterior watershed or borderzone infarcts [35] may produce a picture of bibrachial paralysis with intact motor functioning of the legs (man-in-the-barrel syndrome).

Focal, acute, brachial, crural (one or both extremities) paralysis may be the expression of a spinal cord lesion [1].

Associated symptoms

The association of a sensory deficit with a pure motor deficit constitutes the so-called sensorimotor syndrome, also known as sensorimotor stroke, which is presented as a complete (faciobrachiocrural) or incomplete pyramidal syndrome associated with a complete or partial sensory deficit of the same side of the body [36]. Sensorimotor stroke is the lacunar syndrome that is most often caused by non-lacunar infarcts (Table 1.2) [20]. In a clinical series of lacunar stroke patients, sensorimotor stroke was caused by a symptomatic intracranial small vessel disease in 69.5% of cases. However, other stroke subtypes were found in 30.5% of cases, which is a higher percentage than that observed in other lacunar syndromes (Figure 1.5).

Ipsilateral visual alterations due to ocular ischemia (retinal or anterior) plus contralateral hemiparesis (optopyramidal syndrome) or hemispheric infarction (optocerebral syndrome) is suggestive of internal carotid artery occlusion [3].

The location of brainstem infarctions is highly reliable if motor deficits are associated with signs of nuclear involvement. Crossed brainstem syndromes, well known with eponyms, are characterized by palsy of one of the 12 cranial nerve pairs associated with a contralateral neurological deficit due to involvement of the neurological long tracts (mainly motor or sensory) [3,37,38]. In these cases, the involved cranial nerve suggests the level of the lesion in the brainstem.

The most frequent syndromes due to midbrain lesions [2,37,38] are the following: Weber's syndrome associated with third cranial nerve palsy and contralateral pyramidal deficit; Claude's syndrome with third cranial nerve palsy and
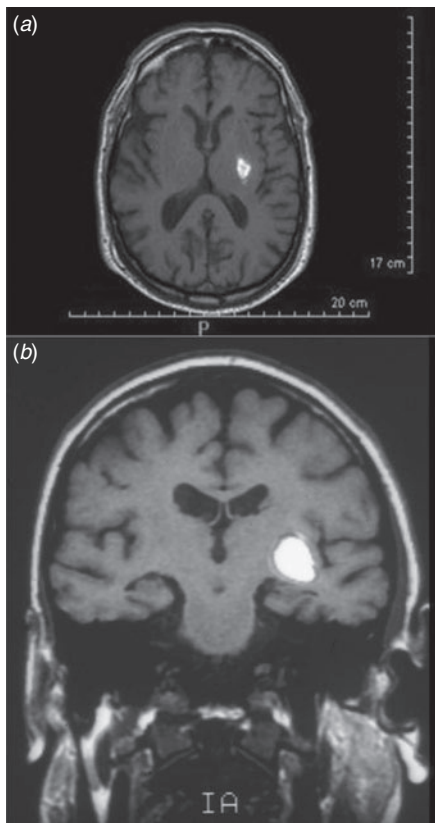
Figure 1.5. Hyperintensity on T1-weighted spin-echo sequences compatible with capsular hemorrhage in a patient with pure motor hemiparesis (a) and a hemorrhage in the basal ganglia in a patient with sensorimotor syndrome (b).

contralateral cerebellar ataxia; Benedikt's syndrome with third cranial nerve palsy and choreoathetosis, hemianesthesia or contralateral tremors; and Nothnagel's syndrome with palsy of the third cranial nerve and hemiataxia, ptosis, and paresis of the superior rectus muscle. An upward fixed gaze and pupils or Parinaud syndrome indicates involvement of the quadrigeminal plate.

Examples of involvement of the pons [3,37,38] includes Raymond's syndrome characterized by sixth cranial nerve palsy and contralateral motor deficit; Raymond–Cestan syndrome with gaze palsy and hemiparesis, hemiataxia, and hemihypalgesia; and Foville's syndrome with seventh cranial nerve palsy. Occlusion of pontine paramedian tracts is the anatomical locus for the Millard–Gubler syndrome, which consists of contralateral hemiplegia and deep sensory anesthesia, and ipsilateral paralysis of the sixth and seventh cranial nerves. Internuclear ophthalmoplegia is characterized by impaired horizontal eye movement with weak adduction of the affected eye and abduction nystagmus of the contralateral eye, resulting from a lesion in the medial longitudinal fasciculus in the pons. Seventh cranial nerve palsy with interruption of the medial longitudinal fasciculus causes the one-and-a-half syndrome, characterized by complete palsy of the movements of the ipsilateral eye and failure of adduction of the other eye. The Brissaud–Sicard syndrome is characterized by hemiparesis and contralateral hemifacial spasm resulting from a pontine lesion.

The Wallenberg syndrome, due to involvement of the lateral medullary tegmentum with involvement of the ninth and tenth cranial nerves, and the Babinski–Nageotte syndrome with involvement of the hemimedulla characterized by the lateral medullary syndrome plus a contralateral hemiparesis, are examples of bulbar lesions.

The association of a pyramidal deficit with a cerebellar syndrome causes ataxic hemiparesis syndrome, usually due to a lacunar lesion in the corticopontocerebellar, dentatorubrothalamocortical, or somesthetic propioceptive pathways of the posterior limb of the internal capsule [39–43], or the pons. It has also been reported in lacunar infarcts of other topographies, including the corona radiata and the thalamus. Ataxic hemiparesis includes the simultaneous presence of a pyramidal syndrome (predominantly crural) associated with a homolateral ataxic syndrome; brachiocrural dissymmetry is not justified by the degree of paralysis. Isolated crural paralysis associated with ipsilateral ataxic hemiparesis may be observed occasionally. In some cases, motor symptoms may be accompanied by a transient sensory deficit, the so-called ataxic hemiparesis with hypoesthesia syndrome [44]. Absence of in-hospital mortality and absence of neurological deficit at discharge from the hospital were present in 39% of the patients in a clinical series of 23 patients with ataxic hemiparesis [43]. Ataxic hemiparesis not due to lacunar stroke is found in less than 7% of cases and may be due to non-lacunar infarcts [43–46], cerebral hemorrhages [47], tumors [48], or infections [49].

Agnosia

There are different types of disturbances of self-perception and impairment of body scheme, which may be present in patients with motor weakness. These include asomatognosia, anosognosia, anosodiaphoria, and misoplegia.

Asomatognosia or hemiasomatognosia (from the Greek *a* for without, *somatos* for body, and *gnosis* for awareness) is the lack of awareness of the contralateral hemibody, usually the left side, in lesions of the right posterior parietal lobe. The patient does not recognize a part of the entire left body. Patients with hemiasomatognosia act as if half of their body no longer exists and, in some cases, patients will even deny that half of their body ever existed. It is usually a transient phenomenon. It was described for the first time by Jean Lhermitte [50] and it is usually found in association with other disturbances of body image, such as unilateral spatial agnosia.

Anosognosia (from the Greek *nosos* for disease or defect), a term introduced by Babinski [51], is characterized by unawareness or denial of the motor deficit such as hemiplegia (usually left hemiplegia) due to a lesion of the right parietal lobe.

Anosodiaphoria is a term introduced by McDonald Critchley [52] as a variant of anosognosia in hemiplegia, in which the patient minimizes or seems indifferent to the existence of the handicap [52–54].

Misoplegia, a term coined by McDonald Critchley [55] (from the Greek *misos* for to hate and *plegia* for paralysis) refers to the morbid dislike or hatred of paralyzed limbs in patients with hemiplegia.

6

Hemiparesis and other neurovascular syndromes – other topographies

Strokes in the ACA territory are uncommon (<2% in stroke registries). Hemiparesis predominating in the lower limb (paralysis is usually greatest in the foot but is also severe in the proximal thigh, and often includes the shoulder shrug but spares the hand), is seen mainly in large infarcts in the ACA territory, being associated with involvement of the parasagittal precentral area and supplementary motor cortex rather than the primary motor cortex. Faciobrachial paresis is not caused by corticospinal tract weakness, but by motor neglect caused by damage of medial premotor areas or its connections [56]. Left-sided infarcts can cause mutism, transcortical motor aphasia, and hemiparesis, and occasionally left arm apraxia. Right-sided lesions can cause acute confusional state, hemiparesis, and motor neglect [3,57]. In some cases, the paresis may be proportional and the ACA stroke pattern will be indistinguishable from an MCA pattern [56].

The malignant combined MCA–ACA infarct situation causes a large cerebral lesion and is often complicated within 24 to 96 hours from the onset of cytotoxic edema, which will produce a mass effect. This may result in clinical deterioration and death due to brainstem compression [3].

The predominant clinical finding of posterior cerebral artery (PCA) infarcts are hemianopia or other visual field defects [57–60]. Infarcts of the PCA usually present without weakness. However, hemiparesis can sometimes occur, resulting from infarction of the cerebral peduncle (peduncular perforators and anterior circumflex arteries) [60]. Posterior cerebral artery infarcts are commonly due to cardiac embolism [61].

In the vertebrobasilar circulation [62], the most common form of acute multiple brain infarcts is the top-of-the-basilar syndrome due to embolism of the junction of the basilar and the PCA, producing occipital, thalamic, and midbrain (or midbrain–superior cerebellar) lesions. These lesions can be related to cardioembolism or artery-to-artery embolism.

In cerebellar infarcts, a motor deficit suggests a brainstem lesion or compression of motor fibers by a mass effect due to edema. Descompressive surgery is life-saving in pseudotumoral cerebellar infarcts or hemorrhages [3,62].

Pontine infarcts can have five main clinical patterns [3,62,63]: ventromedial, ventrolateral, tegmental, bilateral, and unilateral multiple infarct syndromes. Ventromedial infarcts usually are large infarcts that cause severe faciobrachiocrural hemiparesis with or without ataxia and dysarthria, while ventrolateral pontine infarcts usually are small infarcts that cause slight motor dysfunction corresponding to a lacunar syndrome. Caudal or middle ventromedial pontine infarcts correlate with severe hemiparesis, whereas lesions of similar size located in the rostral pons usually have minimal or no limb weakness. A mild facial palsy, much more commonly homolateral to the motor deficit, is often present due to damage of supranuclear fibers at the ventrotegmental junction of the upper or middle pons.

Medullary infarcts can be medial, lateral, or combined [3,62]. In medial medullary infarcts, the motor deficit is usually contralateral and more pronounced in the upper extremity and in the distal portion of the limbs. However, medial medullary infarcts can show four major clinical patterns: (i) Déjérine's syndrome (contralateral hemiparesis, pain, and thermal sensory loss) plus ipsilateral lingual palsy, (ii) sensorimotor stroke without lingual palsy, (iii) hemiparesis often combined with nystagmus, and (iv) tetraparesis caused by bilateral pyramid infarcts. Lateral infarcts produce a Wallenberg's syndrome. Combined or hemimedullary infarcts cause a Babinski–Nageotte syndrome. They are usually secondary to occlusion of the ipsilateral intracranial vertebral artery.

Recurrent strokes may cause a pseudobulbar syndrome, defined by the triad of Thurel of dysarthria, dysphagia (mainly to liquids), and mimic disturbances (laugh or spasmodic cry) [64]. Moreover, it is associated with a peculiar gait (*marche à petits pas*), gait apraxia, and involuntary micturitional urgency. Disorders of higher cerebral function (subcortical-type dementia) are also frequent [65]. There are three anatomoclinical forms of the pseudobulbar syndrome: (i) the corticosubcortical form of Foix–Chavany–Marie or biopercular syndrome, (ii) the pontocerebellar form, and (iii) the striata or central form, which is the more frequent and is usually due to multiple and disseminated lacunar infarcts (corresponding to the lacunar state of Pierre Marie) [19,66]. Early diagnosis and treatment of hypertension and cardiovascular risk factors, as well as the use of platelet antiaggregant drugs for the secondary prevention of brain ischemia, have been determinants for the low occurrence of the classical lacunar status to date.

Prognosis

Motor impairment after stroke has been related to lesion site and size, amount of previous lesion burden, and other factors like age and comorbidities. Recovery after stroke is related to plastic changes in the cerebral cortex. The integrity of all motor tracts, with the pyramidal tract as the main descending fiber bundle, but also the corticorubrospinal and corticoreticulospinal systems, appears to account for stroke recovery in a recent in vivo diffusion tensor imaging (DTI) study in chronic stroke patients [67]. Diffusion tensor imaging studies can provide non-invasive in vivo information about the integrity of cerebral white matter tracts [68]. In a subset of young adults of less than 45 years of age, the clinical prognosis of acute stroke is better. In lacunar stroke, the short-functional prognosis of dysarthria–clumsy hand is excellent [27]. Generally, when the motor or sensory deficit is complete (affecting the face, arm, and leg), the prognosis is worse than in cases with incomplete deficits [19]. The size of the cerebrovascular lesion on CT or MRI is usually correlated with outcome [3,4]. Motor weakness is an important clinical feature that is significantly associated with ischemic stroke (Table 1.3) and early death in MCA ischemic stroke (Table 1.4) [57,69]. In a recent study,

Table 1.3. Variables associated with ischemic stroke caused by anterior cerebral artery, middle cerebral artery, and posterior cerebral artery infarction [57]

Variable	Odds ratio (95% confidence interval)	P
ACA versus MCA infarctions		
Model based on demographics, vascular risk factors, and clinical variables		
Speech disturbances (dysarthria, aphasia)	0.48 (0.27–0.85)	0.012
Altered consciousness	0.31 (0.11–0.88)	0.028
ACA versus PCA infarctions		
Model based on demographics, vascular risk factors, clinical features, and topographic and etiological variables		
Motor deficit	9.11 (3.8–21.8)	0.0001
Cardioembolism	2.49 (1.21–5.14)	0.013
Sensory deficit	0.35 (0.17–0.74)	0.006

ACA, anterior cerebral artery; MCA, middle cerebral artery; PCA, posterior cerebral artery.

Table 1.4. Variables independently associated with in-hospital mortality in cerebral infarctions of the middle cerebral artery [69]

Variable	Odds ratio (95% confidence interval)	P
Model based on demographics, vascular risk factors, and clinical variables		
Early seizures	4.49 (1.77–11.40)	0.002
Age >85 years	2.61 (1.88–2.60)	0.0001
Atrial fibrillation	2.57 (1.89–3.49)	0.0001
Motor weakness	2.55 (1.40–4.66)	0.002
Heart failure	2.33 (1.43–3.80)	0.0001
Sensory deficit	2.29 (1.68–3.12)	0.0001

functional outcome of the affected proximal dominant paresis was better compared with the distal paresis group [11].

Other types of motor weakness: non-pyramidal paresis and topography of the lesion

Frontal lobe

Lesions of the premotor areas in the frontal lobe may cause a global reduction of the motor activity of the contralateral hemibody, with decreased reaction against pain stimuli, so-called motor neglect [1].

There may also be a disturbance of voluntary movements, expressed not by the inability to make a certain gesture, but because of the difficulties in processing, such as delay in onset, difficulty in performing rapid movements, and stopping the movement in the course of the gesture. This difficulty determines the phenomenon of motor perseveration, consisting of the difficulty or impossibility of making one gesture motor activity after another, with persistent repetition of the first motor response. These motor deficits are often accompanied by other contralateral signs, such as the grasp reflex, increased muscular tone, or resistance to movement during manipulation of a limb [1]. Some patients also show abulia – a decrease in spontaneous speech and behavior, prolonged latency in responding to queries or requests for action, and short terse replies.

Parietal lobe

Motor deficits secondary to lesions in the parietal lobe are related to disturbances of sensorimotor integration produced by loss of the somesthetic afferent input. Motor deficits in the form of hemiplegia or monoplegia without true paralysis may be observed in the so-called afferential paralysis or retro-Rolandic form of motor neglect [1].

Pseudoincoordination or parietal ataxia secondary to a proprioceptive sensory disturbance of the parietal cortex causes a difficulty or inability to perform the different actions of a voluntary movement.

Lesions of the parietal lobe may produce disturbances of symbolic functions, with alteration of pain reactivity. In the case of involvement of the minor or nondominant hemisphere, pain hemiagnosia with a decrease in reaction to painful stimuli applied to the left half of the body may be observed. In the case of lesions in the major or dominant hemisphere, the patient may present with pain asymbolia, in which pain in the contralateral hemibody is perceived, but does not cause suffering [1].

Cerebellum

The cerebellum participates in the regulation and control of movement, both in the adaptation of postures and in voluntary movements. For this reason, lesions of the cerebellum may present non-pyramidal motor disturbances in the same side as the lesion [1].

Asynergy refers to defective coordination or inability to perform, in time and space, elemental movements involved in a complex movement action.

The adiadochokinesis is the loss of diadochokinesis, which is the ability to perform rapid alternating movements.

If the lesion causing these disturbances is acute and intense due to the extension of the cerebellar lesion, cerebellar hemiparesis may even be observed.

Extrapyramidal system

Focal movement disturbances secondary to an extrapyramidal condition, with hypokinesis and rigidity, with acute or sub-acute onset, is exceptional (acute parkinsonism) [1,70].

Acute hydrocephalus, intoxication (carbon monoxide, ethanol, methanol), infection, or drugs (neuroleptics) may cause an acute extrapyramidal syndrome, usually bilateral. Cerebral infarction or intracerebral hemorrhage in the substantia nigra is associated with other clinical manifestations associated with parkinsonism.

An incipient parkinsonian syndrome, not previously diagnosed, in a patient of advanced age presenting with acute focal motor deficit, with rigidity and hypokinesia, may be secondary to an extrapyramidal lesion. This may occur when the onset is associated with a metabolic, infectious, toxic, or pharmacological disturbance.

Motor deficit with rigidity of malignant catatonia is bilateral. However, in the prodromic phase, speech disturbances such as autism, echolalia, or echopraxia may be confounded with a focal hemispheric lesion.

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9

Section 1: Clinical manifestations

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