Chapter 2
Presenting signs and symptoms in brain tumors

AGUSTI ALENTORN1,2, KHÉ HOANG-XUAN1,2,3* AND TOM MIKKELSEN3,4
1Department of Neurology 2, Groupe Hospitalier Pitié-Salpêtrière, Paris, France
2Inserm Experimental Neuro-Oncology Laboratory, Institut du Cerveau et de la Moelle, Université Pierre et Marie Curie, Paris, France
3Hermelin Brain Tumor Center and Departments of Neurology and Neurosurgery, Henry Ford Hospital, Detroit, MI, USA
4Wayne State University, Detroit, MI, USA

Abstract
Focal symptoms and signs occurring during brain tumor clinical presentation are dependent on a number of factors. Location and rate of growth are the most critical, followed by overall lesion size and nature, whether infiltrating or causing the displacement of neural structures, but also the presence or extent of associated pathology, including edema, hemorrhage, vascular compromise, and cerebrospinal fluid obstruction. Mechanisms of presenting symptomatology can be divided into tumor and peritumoral factors. Tumor factors include histology, for example, in that seizures are common in patients with certain low-grade gliomas. Peritumoral factors, including regional hypoxia and ionic changes in the peritumoral zone, may influence neuronal activity and extracellular glutamate may be associated with neuronal hyperexcitability. Blood–brain barrier breakdown may predispose to seizure and localized neuronal dysfunction. Finally, signs and symptoms in brain tumors can be generalized, associated with increased intracranial brain pressure, but can also be localized, based on the involvement of the major structures of the central nervous system.

INTRODUCTION
The era of sophisticated and ubiquitous neuroimaging has made many of the clinical distinctions regarding differential diagnosis and localization by semiology rather moot. A detailed investigation regarding the specific etiology of intracranial mass lesions is not well informed by mode of clinical presentation, but much more on epidemiologic and imaging features rather than clinical symptomatology.

A mixture of mechanisms causes the symptoms and signs and their combination produces the clinical syndromes observed in patients (DeAngelis et al., 2002). Jaeckle (1998) reported in the last update to this volume that much of the localizing symptomatology of brain tumors is not specifically tied to specific tumor types, but rather by location within the nervous system. Tumor can damage neural tissue or displace it by compression, leading to focal symptoms. Direct invasion of the tumor typically occurs in infiltrating gliomas or lymphomas, whereas meningiomas and metastases displace brain tissue. The disruption of the blood–brain barrier by the tumor leads to vasogenic edema that is probably one of the main causes of clinical impairment. Edema favors an increased mass effect and thus further compression of the surrounding brain. Furthermore, presenting symptomatology can be often attributed to more generalized symptoms and signs, including headache, altered mental status, seizure and symptoms of increased intracranial pressure, including papilledema, nausea, and vomiting.

As detailed elsewhere, there are also detailed descriptions of the evolution of symptoms associated with various herniation syndromes, but again, these are not specific to tumor, but can be seen with any intracranial mass lesion. As a result, these are not necessarily helpful

*Correspondence to: Professor Khé Hoang-Xuan, Service de Neurologie – 2 2, Groupe Hospitalier Pitié-Salpêtrière, Paris, France. Tel: +33-42-16-03-81, Fax: +33-42-16-04-18, E-mail: khe.hoang-xuan@psl.aphp.fr
for diagnosis, whereas neuroimaging can quickly distinguish most responsible lesions.

Mechanisms of presenting symptomatology can be divided into tumor and peritumoral factors. Tumor factors include histology, for example, in that seizures are common in patients with certain low-grade gliomas. Rapidly growing high-grade tumors may cause more widespread tissue destruction, whereas more indolent tumors may cause partial deafferentation of cortical neurons, with resulting denervation hypersensitivity and hence a more epileptogenic microenvironment.

Tumor location is obviously critical regarding symptomatology, but certain locations are closely related to certain histologies and can suggest the relative likelihood of certain tumors. Glioneuronal tumors and astrocytomas appear to favor the temporal lobes and oligodendrogliomas may more likely be located in frontal lobes. It is not clear whether these location correlations have any neurodevelopmental or mechanistic basis.

Peritumoral factors, including regional hypoxia blood–brain barrier breakdown, may predispose to seizure and localized neuronal dysfunction. Ionic changes in the peritumoral zone may influence neuronal activity and extracellular glutamate may be associated with neuronal hyperexcitability.

**GENERALIZED SYMPTOMS AND SIGNS**

**Headache**

Prospective studies and retrospective case series have attempted to assess the risk of intracranial mass or neoplasm in patients presenting with headache. Interestingly, it is important to note that the prevalence of brain imaging abnormalities on magnetic resonance imaging is low in patients without headache. A meta-analysis of 16 studies (*n* = 19 559) showed a prevalence of asymptomatic brain neoplasm of 0.7% (95% confidence interval, 0.47–0.98%) (Morris et al., 2009). The American Academy of Neurology has published guidelines for red flags (features suggesting concern), which include abnormal neurologic examination, headache worsened by Valsalva maneuver, headache causing awakening from sleep, new headache in the older population, progressively worsening headache, atypical headache features, or patients who do not fulfill the strict definition of migraine (Schaefer et al., 2007).

Although headache is the assumed “first sign” of brain tumor, it is relatively rare as a sole presenting complaint in patients with tumor. Vázquez-Barquero et al. (1994) performed a prospective study over the course of 2 years in 183 patients with brain tumor, and identified 15 patients (8%) with headache as the only complaint on presentation. Although only 8% had presented with headache as the only complaint, at the time of tumor diagnosis, 64% complained of headache, with only 1 patient having headache as the sole complaint. In those presenting with headache as an initial symptom, posterior fossa location and hydrocephalus were more frequent. Primary tumor versus metastatic tumor prevalence among headache sufferers was 60% and 40%, respectively, with no statistically significant difference in prevalence between any histopathologic diagnostic groups (Vázquez-Barquero et al., 1994).

The “classic” presentation of tumor headache refers to head pain that is present on awakening, with a dull, constant character and of severe intensity, with nausea and vomiting (Forsyth and Posner, 1993). Pfund et al. (1999) noted several nonclassic findings in their prospective study. Morning headache occurred in only 31.8% of patients, and headache was daily in only 10.6% of patients. Pain was more likely intermittent (88.4%) than constant, with typical headache duration of hours (81.8%). While these characteristics were not typical for classic presentation, other more classic features included throbbing pain in 63%, association with nausea and vomiting in 59.6%, and moderate to severe intensity in 90% of patients. Headaches were progressive in those patients with glioblastoma, astrocytoma, and metastatic tumors. Hemispheric tumors tended to be ipsilateral, and infratentorial tumors caused frontal/temporal/parietal pain (Pfund et al., 1999).

Valentinis et al. (2010) used International Classification of Headache Disorders, second edition (ICHD-II) criteria to diagnose headache types. Episodic tension-type headache was present in 23.5% of patients, and episodic migraine without aura occurred in 13.3%. Among those with tension-type headache, 19 of 23 patients had new-onset headache, with 9 having atypical features. Among the migraine type, 8 of 13 patients reported a change in pre-existing headache. Atypical features were present in 92.3%, including worsening with Valsalva, positional headache, or unresponsiveness to medication. Morning headache was present in 25.5%, with 46.9% of patients reporting headache awakening them from sleep. Headaches more typically occurred 1–3 days per week (40.8%) and 4–6 days per week (23.5%). Pain was bilateral in 61.2% and side-locked in 29.6%. Laterality of headache was predictive of tumor location, with 24 of 29 patients with side-locked headache occurring ipsilateral to the tumor. Of 60 patients with strict bilateral headache, 53.3% had bi-hemispheric tumor and 25% had midline tumors. Pain was daily in 8.2% of cases, with constant pain in 13.3%. Pain was of pressing/tightening quality in 60.2% of patients and pulsatile in 33.7%. Headache was more often moderate, with severe headache in 35.7%. Duration was 30 minutes to 4 hours in 41.8% and 4–72 hours in 35.7% of patients. Nausea was present in 34.7% and vomiting in 23.5%. These findings differ from
Nausea and vomiting

Nausea and vomiting occur when the chemotactic trigger zone in the area postrema, located on the floor of the fourth ventricle, is stimulated. Vomiting usually indicates raised intracranial pressure, but it can also occur because of direct compression of the vomiting center by posterior fossa tumors such as medulloblastoma or ependymomas of the fourth ventricle. It can also occur in the absence of elevated intracranial pressure in brainstem tumors involving the nucleus solitarius. Acute headache followed by an episode of vomiting suggests increased intracranial pressure. Projectile vomiting, usually seen in posterior fossa tumors in children, is uncommon in adults. Ictus emeticus or vomiting as an ictal phenomenon can, rarely, be a manifestation of epilepsy in tumors involving the insula and mesial temporal lobe. It has been reported with temporal-lobe astrocytoma (Chen et al., 1999) and mesial temporal glioma (Schäuble et al., 2002).

Altered mental status

Mental and cognitive abnormalities may be specific or nonspecific. Specific findings include aphasia, agnosia, abulia, alexia, or apraxia. These symptoms have localizing value but are often mistaken for global cognitive impairment.

Nonspecific mental changes are among the most common symptoms and are often subtle, particularly early in the disease course. Irritability, change in personality, emotional lability, forgetfulness, lack of enthusiasm or spontaneity, and slowed response, progressing gradually to apathy and lethargy, are seen in about 16–34% of patients (Weitzner, 1999). These symptoms do not reflect tumor in a specific area of the brain, but, when present, are suggestive of tumors in deep structures affecting thalamocortical fibers, corpus callosum, reticular formation, and, occasionally, the frontal lobes. Variations in behavior can be seen according to which area of the frontal lobe is involved; for example, apathy and abulia are seen in tumors involving the dorsolateral frontal lobe, whereas orbitofrontal tumors lead to disinhibition and impulsive behavior. Sometimes withdrawal and apathy are confused with depression, even when the patient denies feeling sad or depressed. Aggressive and impulsive behavior can be seen with lesions involving the amygdala. Occasionally, temporal-lobe seizures present with behavioral disturbances. Malamud (1967) has described outbursts of rage in patients with temporal-lobe gliomas. Peduncular hallucinosis can occur with midbrain tumors, and has also been described in posterior fossa tumors compressing the brainstem (Nadvi and van Dellen, 1994; Leiva-Santana et al., 1999). Behavior and personality changes predominate as the presenting complaints, particularly in primary central nervous system lymphoma. This is due to its predilection for the frontal lobes and a 40% incidence of multiple lesions at diagnosis. Raised intracranial pressure can present with confusion, disorientation, lethargy, and coma.

Papilledema

Papilledema is an indicator of increased intracranial pressure; it is now rarely seen in patients at the time of presentation of a brain tumor. This is due to the widespread availability of modern neuroimaging, which is almost always performed before the tumor progresses far enough to cause papilledema. In its mildest form, or in acute cases, it may not lead to a change in visual acuity; enlargement of the blind spot may be the only finding on examination. Like headache, papilledema is seen more often in children and young adults; this is probably because older adults have more room for tumor expansion due to brain atrophy, or because, in older adults, optic nerve sheath fibrosis does not allow the pressure to be transmitted to the disc. Papilledema due to raised intracranial pressure is usually bilateral, although it may not be symmetric. Certain chronic conditions, such as a frontal-lobe tumor or olfactory groove meningioma, may, rarely, lead to the Foster–Kennedy syndrome, in which there is optic atrophy on the side of the tumor due to chronic local compression, and papilledema on the contralateral side due to increased intracranial pressure.

Seizure

Seizures are a frequent symptom in patients with a brain tumor. The incidence varies between 30% and 100% and depends on the tumor type, with slow-growing tumor being the most epileptogenic (van Breemen et al., 2007). Low-grade tumors may be more refractory than higher-grade lesions (Hildebrand et al., 2005). Seizures that develop late in the clinical course are typically more responsive to treatment than those that occur early (Glantz et al., 1996). Seizures that are initially controlled but return at the time of tumor recurrence are relatively refractory to treatment (Whittle and Beaumont, 1995). The prognosis for complete seizure control in patients...
with tumor-associated epilepsy is relatively poor. Indeed, more than 50% of patients with gliomas experience recurrent seizures during the course of their disease, while 11% of patients with brain metastasis and 19% of patients with neoplastic meningitis suffer recurrent seizures during their substantially shorter lifetimes (Hildebrand et al., 2005). Tumor-associated epilepsy can be classified under “symptomatic epilepsy,” which is defined as “the development of epilepsy caused by an identifiable injury or lesion” (Engel et al., 2007). It is likely that in tumor-related epilepsy multiple mechanisms are involved, including tumor-related factors (histologic type, location), environment-related factors, and functional changes. Furthermore, several hypotheses regarding the pathophysiology of tumor-associated epilepsy have been proposed, such as blood–brain barrier disruption, pH imbalance, and metabolic changes due to vascular disturbance, cytokine and glutamate concentrations, localized immunologic interactions, and hypoxia. Such hypotheses have been previously summarized by Beaumont and Whittle (2000) and Shamji et al. (2009).

**CLINICAL FEATURES OF INCREASED INTRACRANIAL PRESSURE**

**CSF obstruction**

The classic presentation of cerebrospinal fluid (CSF) obstruction was described by Dandy in 1922 in a case of third ventricular colloid cyst with intermittent obstruction and positional recumbent headache. Nevertheless, it should be noted that a prospective study of 105 colloid cysts of the third ventricle found only 2 patients with positional headache (Desai et al., 2002). Generalized intermittent headache was present in 92.3% of patients, with associated ataxia (25.7%), blurred vision (20%), papilledema (72.4%), urinary incontinence (17.1%), memory deficits (9.5%), seizure (7.6%), coma (3.8%), hemiparesis (2.9%), and normal-pressure hydrocephalus (3.8%). While such headaches are resolved completely with surgery, the consequences of delay to surgery can be devastating. The authors noted that all 4 patients presenting with coma died before surgery could be performed (Desai et al., 2002). Tumors in the third or fourth ventricles, such as colloid cysts, ependymomas, choroid plexus papillomas, and meningiomas, can obstruct CSF outflow, resulting in hydrocephalus. In these situations, hydrocephalus may be the presenting feature. Tumor in the leptomeninges can lead to communicating hydrocephalus. Accordingly, hydrocephalus and raised intracranial pressure may occur as a delayed complication of hemispheric tumors such as glioblastoma and metastases.

**Herniation syndromes**

Brain tumors cause shift or herniation of brain tissue from one compartment to another. The most important clinical consequence of a shift is a depressed level of consciousness. False localizing signs may be seen, most commonly in patients with a herniation syndrome and raised intracranial pressure. A herniation syndrome is a rare presentation of an undiagnosed brain tumor. It is much more likely to be seen in a patient known to have an intracranial mass that has progressed despite treatment, usually late in the course of a brain tumor.

There are five common herniation syndromes (Posner et al., 2007):

1. **Subfalcine herniation:** The cingulate gyrus is pushed under the falx cerebri, leading to compression of both anterior frontal lobes or occlusion of an anterior cerebral artery with subsequent frontal-lobe infarction.
2. **Uncal herniation occurs when a mass lesion causes the uncus of the temporal lobe to herniate through the tentorium cerebelli.** The key clinical sign of uncal herniation is ipsilateral oculomotor nerve palsy with a fixed and dilated pupil due to compression by the medial temporal lobe. Typically, a contralateral hemiparesis is also seen, but a false localizing sign of uncal herniation is an ipsilateral hemiparesis due to displacement of the brainstem to the opposite side causing compression of the contralateral cerebral peduncle against the tentorium (Kernohan notch). Additionally, unilateral or bilateral posterior cerebral artery occlusion can occur at the tentorial notch, leading to a homonymous hemianopsia or cortical blindness.
3. **Central herniation is due to pressure from a mass in the diencephalon and may lead to coma.** Rostrocaudal shifts compress the brainstem, compromising its vascular supply and leading to Duret hemorrhages.
4. **Tonsillar herniation occurs when the cerebellar tonsils herniate through the foramen magnum; this may cause obstruction of the fourth ventricular outflow, and compression of the medulla with sudden death.**
5. **Upward brainstem herniation is seen in patients with posterior fossa tumors, where the superior surface of the vermis and midbrain are pushed upward, compressing the dorsal mesencephalon and cerebral aqueduct.** Dorsal midbrain compression leads to impairment.

**LOCALIZING SYMPTOMS AND SIGNS OF INTRACRANIAL NEOPLASMS**

Localizing symptoms and signs of intracranial neoplasms can differ based on the relative preponderance
of lesions responsible, but these are typically only relatively specific. Patterns of growth, either bulk growth or infiltrative growth, may also impact the evolution of clinical symptoms and signs (Biller et al., 2011).

Stroke-like presentation may be due to intratumoral hemorrhage, but rapid symptom onset and evolution may also be seen in evolving mass effect even without sudden intratumor hemorrhage, especially with compressive and herniation syndromes (Posner et al., 2007). Intratumoral hemorrhages may be seen in any primary or metastatic brain tumor; metastatic melanoma, choriocarcinoma, and renal or thyroid cancer are most common. Primary brain tumors, including glioblastoma and oligodendroglioma, may be associated with intratumor hemorrhage, especially in those predisposed owing to therapeutic anticoagulation.

Invasion from the skull

Skull base neoplasms, be they derived from the bony skull, dura, or destructive tumors metastatic to these sites from hematogenous or direct extension, may result in local infiltration, with localized pain and/or localized dysfunction, either of cranial nerves, orbital contents or, more posteriorly, brainstem and long tracts.

Retro-orbital tumors, again either of bony origin or the result of local infiltration, can involve the cranial nerves, either at the orbital apex or adjacent cavernous sinus, with disorders of vision, ocular motility, or facial sensation.

Middle cranial fossa tumors can present with symptomatology related to specific cranial nerves as they exit the skull base, in addition to their mass effect on parenchymal structures, which will be covered below.

Owing to the proximity of the clivus to lower cranial nerves and the long tracts of the brainstem, masses in this area will often present with a combination of lower cranial nerve involvement, in addition to long-tract or pyramidal signs, including pyramidal weakness, hypertonus, and hyperreflexia.

Similarly, posterior fossa extra-axial masses, such as meningiomas and skull base metastases, may present with a similar combination of signs, but with additional compromise of cerebellar function and, possibly, the superimposition of mass effect on the CSF pathways and possible CSF obstruction. Parenchymal cerebellar hemisphere masses may present with cerebellar hemispheric signs, including dysmetria or tremor, in addition to pyramidal weakness, depending on mass effect or involvement of the adjacent pons or midbrain or projections. This may be sudden, especially with parenchymal tumors following hemorrhagic conversion, for example, as seen in certain metastatic tumors.

Masses in the brainstem, depending on their symmetry and level, will often present with a combination of ipsilateral cranial nerve symptomatology in addition to long-tract or pyramidal signs, classically on the contralateral side.

Frontal-lobe tumors

General features

Frontal-lobe tumors may be subtle and relatively nonspecific, but progressive symptoms may raise suspicion for mass lesion or the superimposition of symptoms of elevated intracranial pressure or seizure may prompt investigation.

Higher cognitive function, especially personality change, flattening of the affect, and cognitive dysfunction usually suggest a deeper lesion, often bilateral. As disturbances progress, an apathetic state may occur, sometimes only noticeable in retrospect, with indifference to incontinence and lack of insight.

Gait and posture disorders, resulting in gait instability, gait initiation signs, and symptoms, may often be seen with more indolent masses. Gait apraxia has been extensively described, with patients having difficulty initiating gait or even other motor movements. Gaze apraxia may occur as a result of the involvement of the frontal eye fields. Other more complex disorders of organized movement, such as ideomotor apraxia, have also been described in the classic literature.

Disturbances of speech, depending on specific nature of the deficit, suggest dysfunction in the dominant hemisphere. Historically, anatomic localization was inferred depending on the specific nature of a speech deficit, but functional imaging has clearly demonstrated the widely differing localization of speech tasks patient to patient. Still, the classic Broca’s aphasia often localizes to the posterior inferior frontal gyrus, and is associated with expressive difficulty and naming and repetition deficits. Paraphasias may be common and, typically, receptive speech is usually retained.

Motor weakness suggests lesions in the posterior extent of the frontal lobe and, if limited in size, may predominantly affect motor function in the lower extremity if superior and medial, or the upper extremity and face if more lateral.

Temporal-lobe tumors

General features

Seizures

The temporal-lobe location of mass lesions may predispose to seizures more than other locations in the brain.
Typically they may have partial onset, with partial complex seizure phenomena particularly common.

**Visual deficits**

Visual field defects are also particularly common with temporal-lobe mass lesions, owing to the disruption of the optic radiations; Meyer’s loop in particular is associated with homonymous upper quadrantic defects.

**Dysphasia**

Expressive speech deficits are also seen with certain temporal-lobe mass lesions. While extensive descriptive detail has been applied to the study of these patients, enough variability in symptomatology and functional localization of speech with patient-to-patient variability limits the precise localization of lesions based on clinical features alone, especially in the era of detailed neuroimaging.

**Cognitive and memory dysfunction**

**Parietal-lobe tumors**

**SEIZURE**

Seizures with focal onset, with partial motor or sensory features, are often characteristic of parietal-lobe masses, in particular those in the region of the motor cortex. There may be early secondary generalization.

**Sensory abnormalities**

Primary sensory dysfunction or more complex or associative symptomatology may occur if the lesion is in the parietal lobe. Dominant-hemisphere lesions are classically associated with agraphesthesia, left–right confusion, and finger agnosia, whereas nondominant-hemisphere lesions are classically associated with neglect phenomena, which may be complex.

**Visual disturbances**

Optic radiations extending posteriorly through the parietal lobe are associated with inferior quadrant defects, although, in our experience, these are seldom as discrete as classically described.

**Dyspraxia**

Various disorders of voluntary action can involve body and spatial disturbances and are associated with parietal-lobe mass lesions.

**Dysphasia**

Posterior temporal/inferior parietal lesions are associated with receptive speech disorders. Parietal involvement may be associated with more complex associative features than classical Wernicke’s aphasia.

**Occipital tumors**

Visual defects are the *sine qua non* for occipital mass lesions, with homonymous hemifield involvement typical. Macular sparing is also typical for occipital lesions, although not universal.

While seizures may occur with cortical lesions anywhere in the brain, occipital lesions are relatively less likely to produce them. Local phenomena such as visual hallucinations may be seen.

Dysfunction of oculomotor tracking is also classically described, including optokinetic nystagmus, but not typically utilized for localizing purposes today.

Color anomia and visual anomia, especially the phenomenon of facial agnosia or aprosopagnosia, are rare.

**Diencephalic tumors**

Diencephalic tumors, in particular due to their mass effect on the third ventricle, may be associated with CSF obstruction at the foramen of Monroe and hydrocephalus.

**Pineal-region tumors**

Pineal-region tumors may present based on either their local mass effect, often associated with CSF obstruction, or pressure on the tectum of the midbrain and oculomotor dysfunction, including the classic Parinaud’s syndrome of vertical-gaze paresis.

**Mesencephalic tumors**

Given the limited size of the brainstem structure and precarious nature of CSF flow pathways, mesencephalic tumors are highly likely to be associated with obstruction of CSF flow. Also, given the specific mesencephalic tectal location of the oculomotor nuclei, disorders of ocular motility are common. As above, involvement of the dorsum from intrinsic lesions may show impairment of vertical gaze, as is seen in pineal extrinsic lesions. Specific disruption of oculomotor function involving the third and fourth cranial nerves is also regularly seen along with combinations of gaze paresis and limitations of specific oculomotor function, as can be seen in the classic one-and-a-half syndrome.
Pontine tumors
Again, given the proximity of cranial nerve and corticospinal fibers responsible for contralateral motor dysfunction, another classic clinical presentation localizing to the pons would be an ipsilateral abducens palsy and contralateral hemiparesis. Other cranial nerves, which may be associated with long-tract signs in the pons, include facial and trigeminal, though, given the extent of the trigeminal sensory nucleus, isolated facial sensory dysfunction is relatively rare.

Cerebellar tumors
Given the proximity of the cerebellum immediately dorsal to the pons and forming the roof of the fourth ventricle, it is no surprise that midline cerebellar lesions may compromise CSF flow and produce hydrocephalus. Lateral cerebellar lesions may also do so, of course, but are typically larger. Lateral cerebellar lesions are also associated with limb ataxias and disrupted coordination, including ocular motor coordination with nystagmus, whereas midline lesions are likely to present with CSF obstruction and more midline ataxia.

Cerebellopontine angle tumors
Masses in the cerebellopontine angle will most often present with involvement of cranial nerves, usually the acoustic, in addition to the extrinsic mass effect of the mass on the adjacent cerebellum. Facial involvement is also often seen, give the immediately adjacent location of the extra-axial facial nerve to the acoustic.

Vestibular schwannoma/acoustic neuroma is the classic presentation, with vertigo, tinnitus, or hearing loss as the presenting symptom.

Medullary tumors
As the brainstem extends to the medulla, lower cranial nerves, including glossopharyngeal, vagus, accessory, or hypoglossal, involvement is seen, but given the compact nature of the brainstem at this level, not usually without long-tract symptoms and signs, which are usually not strictly hemiparesis at this level, but more likely quadriplegic deficits. Given the decussation of the sensory trigeminal pyramids, cranial nerve deficits at this level may be associated with contralateral facial sensory changes.

Spinal cord tumors
Spinal cord tumor presentation is most commonly associated with pain. Pain is the first symptom in >90% of patients presenting with epidural metastasis and occurs less frequently with intradural tumors. Pain occasionally can be absent in adults and more often is absent in childhood. If other neurologic symptoms suggestive of myelopathy are present, without pain, including paraparesis, hypertonus, and hyperreflexia, the clinician should evaluate for spinal cord tumor. These findings may be absent early in the clinical course, especially with extra-axial compressive lesions, including epidural masses and bony collapse from spinal metastasis, and produce spinal shock. Changes in bowel and bladder habits, particularly urinary retention with overflow incontinence, usually occur late in the course of epidural spinal cord compression but are seen in a small percentage of patients at presentation. Sensory-level findings may help localize the specific spinal cord segment involved.

REFERENCES


