3 Neurophysiology and Regional Brain Syndromes

3.1 Neurophysiology

3.1.1 Blood-brain barrier

General information

The passage of water-soluble substances from the blood to the CNS is limited by tight junctions (zonulae occludentes) which are found between cerebral capillary endothelial cells, limiting penetration of the cerebral parenchyma (blood-brain barrier, BBB), as well as between choroid plexus epithelial cells (blood-CSF barrier).[1] A number of specialized mediated transport systems allow transmission of, among other things, glucose and certain amino acids (especially precursors to neurotransmitters).

The efficacy of the BBB is compromised in certain pathological states (e.g., tumor, infection, trauma, stroke, hepatic encephalopathy…), and can also be manipulated pharmacologically (e.g., hypertonic mannitol increases the permeability, whereas steroids reduce the penetration of small hydrophilic molecules).

The BBB is absent in the following areas: circumventricular organs[2] (area postrema, median eminence of the hypothalamus, neurohypophysis (posterior pituitary), pineal gland…) choroid plexus, tuber cinereum, and preoptic recess.

Means of assessing the integrity of the BBB:

- visible dyes: Evan's blue, fluorescein
- radioopaque dyes (imaged with CT scan[3]): iodine (protein-bound contrast agent)
- paramagnetic (imaged on MRI): gadolinium (protein-bound contrast agent)
- microscopic: horseradish peroxidase
- radiolabeled: albumin, sucrose

Cerebral edema and the blood-brain barrier

Two basic types of cerebral edema; diffusion-weighted MRI (p. 243) may be able to differentiate:

1. cytotoxic: BBB is closed, therefore no protein extravasation, therefore no enhancement on CT or MRI. Cells swell then shrink. Classic examples: cell death due to head trauma or stroke
2. vasogenic: BBB is disrupted. Protein (serum) leaks out of vascular system, and therefore may enhance on imaging. Extracellular space (ECS) expands. Cells are stable. Responds to corticosteroids (e.g., dexamethasone). Seen, e.g., surrounding metastatic brain tumor

Cerebral edema related to ischemia may be a combination of the above. BBB is closed initially, but then may open. ECS shrinks then expands. Fluid extravasates late. May cause delayed deterioration following intracerebral hemorrhage (p. 1615)

3.1.2 Language and speech function

Localizing language function

Caveat: Language function cannot be reliably localized on anatomic grounds alone due to individual variability. In order to perform maximal brain resections (e.g., for tumor) while minimizing the risk of aphasia, techniques such as awake intraoperative brain mapping (p. 1735)[4] need to be employed.

Classic model

The model of speech and language function that was accepted for years was that of 2 primary areas, Wernicke's area (Brodmann area 40 and part of 39), which subserved language, and Broca's area (Brodmann area 44), which was considered the "motor speech" area, both located in the dominant hemisphere (▶ Fig. 1.1). These two areas were thought to communicate via the arcuate fasciculus (p. 246).

Lesions in Wernicke's area were classically thought to produce "receptive aphasias," wherein the patient could not understand language. Some of these patients demonstrated "fluent aphasia," in which they generated speech without content. Conversely, patients with lesions in Broca's area would exhibit "expressive aphasia," wherein they could comprehend language, but lacked the motor

ability to generate speech. “Conduction aphasia” was considered to be the result of damage to the arcuate fasciculus.

Dual stream model of language

A model that incorporates current understanding of speech and language[5] (▶ Fig. 3.1).

Region 1: (primary auditory areas) initial processing of language.

The ventral stream flows from Region 1 to Region 2 (anterior and middle temporal lobe) and is involved in speech recognition and lexical concepts.

The dorsal stream maps phonological information onto motor areas (Regions 4 (premotor cortex) & 5 (≈ Broca’s area)).

Regions 3 (≈ Wernicke’s area), 4 & 5 are all involved in dorsal stream processing.

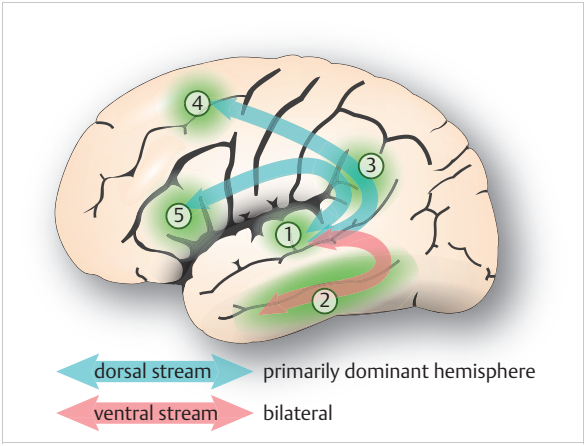

Fig. 3.1 Dual stream model of language function. See text for descriptions of numbered green shaded Regions 1–5.

3.1.3 Babinski sign and Hoffmann sign

Introduction

Although the Babinski sign is regarded as the most famous sign in neurology, there is still disagreement over what constitutes a normal response and when abnormal responses should occur.[6] The following represents one interpretation.

The plantar reflex (PR) (AKA Babinski sign after Joseph François Félix Babinski [1857–1932], a French neurologist of Polish descent) is a primitive reflex, present in infancy, consisting of extension of the great toe in response to a noxious stimulus applied to the foot. The small toes may fan, but this is not a consistent nor clinically important component. The PR disappears usually at ≈ 10 months of age (range: 6 mos to 12 yrs), presumably under inhibitory control as myelination of the CNS occurs, and the normal response then converts to plantarflexion of the great toe. An upper motor neuron (UMN) lesion anywhere along the pyramidal (corticospinal) tract from the motor strip down to ≈ L4 will result in a loss of inhibition, and the PR will be “unmasked” producing extension of the great toe. With such an UMN lesion, there may also be associated flexor synergy resulting in concurrent dorsiflexion of the ankle, and flexion of the knee and hip (AKA triple flexor response) in addition to extension of the great toe.

Neuroanatomy

The afferent limb of the reflex originates in cutaneous receptors restricted to the first sacral dermatome (*S1*) and travels proximally via the tibial nerve. The spinal cord segments involved in the reflex-arc lie within *L4–2*. The efferent limb to the toe extensors travels via the *peroneal nerve*.

Table 3.1 Differential diagnosis of the plantar reflex (PR)

Etiologies
• spinal cord injuries[a] • cervical spinal myelopathy • lesions in motor strip or internal capsule (stroke, tumor, contusion…) • subdural or epidural hematoma • hydranencephaly • toxic-metabolic coma • seizures • trauma • TIAs • hemiplegic migraine • motor neuron disease (ALS)

[a]In spinal cord injuries, the PR may initially be absent during the period of spinal "shock" (p. 1119)

Differential diagnosis

Etiologies

Lesions producing a PR need not be structural, but may be functional and reversible. The roster of possible etiologies is extensive, some are listed in ▶ Table 3.1.

Eliciting the plantar reflex, and variations

The optimal stimulus consists of stimulation of the lateral plantar surface and transverse arch in a single movement lasting 5–6 seconds.[7] Other means for applying noxious stimuli may also elicit the plantar reflex (even outside the S1 dermatome, although these do not produce toe flexion in normals). Described maneuvers include Chaddock (scratch the lateral foot; positive in 3% where plantar stimulation was negative), Schaeffer (pinch the Achilles tendon), Oppenheim (slide knuckles down shin), Gordon (momentarily squeeze lower gastrocnemius), Bing (light pinpricks on dorsolateral foot), Gonda or Stronsky (pull the 4th or 5th toe down and out and allow it to snap back).

Hoffmann's (or Hoffman's or Hoffmann) sign

Attributed to Johann Hoffmann, a German neurologist practicing in the late 1800s. May signify a similar UMN interruption to the upper extremities. Elicited by flicking downward on the nail of the middle or ring finger: a positive (pathologic) response consists of involuntary flexion of the adjacent fingers and/or thumb (may be weakly present in normals).[8] Differs from the plantar reflex since it is monosynaptic (synapse in Rexed lamina IX).

Can sometimes be seen as normal in a young individual with diffusely brisk reflexes & positive jaw jerk, usually symmetric. When present pathologically, represents disinhibition of a C8 reflex, ∴ indicates lesion above C8.

Hoffmann sign was observed in 68% of patients operated on for cervical spondylotic myelopathy.[8] In 11 patients presenting with lumbar symptoms but no myelopathy, a bilateral Hoffman sign was associated with occult cervical spinal cord compression in 10 (91%).[8] The Hoffmann test has a sensitivity of 33–68%, a specificity of 59–78%, a positive predictive value of 26–62% and a negative predictive value of 67–75%.[9]

Conclusion: Hoffmann sign has too low a predictive value for it to be relied upon by itself as a screening tool for, or as an indication of the presence of, myelopathy.[9,10]

3.1.4 Bladder neurophysiology

Central pathways

The primary coordinating center for bladder function resides within the nucleus locus coeruleus of the pons. This center synchronizes bladder contraction with relaxation of the urethral sphincter during voiding.[11]

Voluntary cortical control primarily involves inhibition of the pontine reflex, and originates in the anteromedial portion of the frontal lobes and in the genu of the corpus callosum. In an uninhibited bladder (e.g., infancy), the pontine voiding center functions without cortical inhibition and the detrusor muscle contracts when the bladder reaches a critical capacity. Voluntary suppression from the cortex via the pyramidal tract may contract the external sphincter and may also inhibit detrusor contraction. Cortical lesions in this location → urgency incontinence with inability to suppress the micturition reflex.[12(p 1031)]

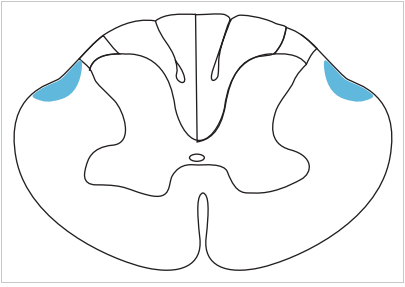

Fig. 3.2 Location of spinal cord bladder efferents in the spinal cord (shaded).

Efferents to the bladder travel in the dorsal portion of the lateral columns of the spinal cord (shaded areas in ▶ Fig. 3.2).

Motor

There are two sphincters that prevent the flow of urine from the bladder: internal (autonomic, involuntary control), and external (striated muscle, voluntary control).

Parasympathetics (PSN)

The detrusor muscle of the bladder contracts and the internal sphincter relaxes under PSN stimulation. PSN preganglionic cell bodies reside in the intermediolateral gray of spinal cord segments *S2–4*. Fibers exit as ventral nerve roots and travel via pelvic splanchnic nerves (nervi erigentes) to terminate on ganglia within the wall of the detrusor muscle in the body and dome of the bladder. These are the target receptors of anticholinergic medications and onabotulinumtoxinA (Botox™).[13]

Sympathetics

Sympathetic cell bodies lie within the intermediolateral gray column of lumbar spinal cord from segments T12—L2. Preganglionic axons pass through the sympathetic chain (without synapsing) to the inferior mesenteric ganglion. Postganglionic fibers pass through the inferior hypogastric plexus to the bladder wall and internal sphincter. Sympathetics heavily innervate the bladder neck and trigone. Stimulation of alpha-1 adrenergic receptors results in bladder neck closure allowing bladder filling and urine storage. Beta-3 adrenergic receptor stimulation results in detrusor smooth muscle relaxation during bladder filling and storage.[14]

Pelvic nerve stimulation → increased sympathetic tone → detrusor relaxation & increased bladder neck tone (allowing a larger volume to be accommodated).

Somatic nerves

Somatic voluntary control descends in the pyramidal tract to synapse on motor nerves in S2–4, and then travels via the pudendal nerve to the external sphincter. This sphincter may be voluntarily contracted, but relaxes reflexly with opening of the internal sphincter at the initiation of micturition. Primarily maintains continence during ↑ vesical pressure (e.g., Valsalva).

Sensory

Bladder wall stretch receptors sense bladder filling and send afferent signals via myelinated A-delta fibers (aid sensation during filling and emptying) and unmyelinated C fibers (sense noxious stimuli, thought to be involved in involuntary detrusor overactivity in neurogenic bladder[15]). These fibers run through the pelvic, pudendal, and hypogastric nerves to spinal cord segments T10–2 & S2–4. Fibers ascend primarily in the spinothalamic tract.

Urinary bladder dysfunction

General information

Bladder management is vital to protect the kidneys from obstruction and subsequent loss of renal function.

Neurogenic bladder: bladder dysfunction due to lesions in the central or peripheral nervous systems. Clinical manifestations differ based on the location of the lesion.

- detrusor hyperreflexia (detrusor overactivity (DO)): involuntary contraction of the detrusor muscle → sensation of urgency & possible urge incontinence
- detrusor-sphincter dyssynergia (DSD): detrusor contraction with inappropriate activation of the external urethral sphincters
- detrusor areflexia: loss of bladder tone → inability to contract sufficient for micturition[16]

Specific injuries affecting the bladder

Classic descriptions of location of lesions are described below; however, over 50% of patients do not have classic presentation or symptoms.

1. supraspinal (lesions above the brainstem): loss of centrally mediated inhibition of the pontine voiding reflex. Voluntary inhibition of micturition is lost. Coordination of detrusor filling and contraction with smooth and striated urinary sphincters is intact, allowing maintenance of normal bladder pressures with low risk of high pressure renal damage. Patients have DO without DSD. Detrusor hypertrophy is less pronounced. Symptoms: urinary frequency or urgency, urgency incontinence, and nocturia.[11] If sensory pathways are interrupted, unconscious incontinence occurs (insensate incontinence AKA incontinence of the unawares type). Voluntary bladder emptying may be maintained and timed voiding together with anticholinergic medications (see below) are used in management. Areflexia may sometimes occur
2. complete (or near complete) spinal cord lesions:
   a) suprasacral (lesion *above* the S2 spinal *cord* level, which is ≈ T12/L1 vertebral body level in an adult): loss of innervation to the pontine micturition center results in reflexive voiding modulated by the sacral voiding center (located in the conus medullaris).[17]
   Etiologies: spinal cord injuries, tumors, transverse myelitis.
      - initially following spinal cord injury, there may be spinal shock. During spinal shock (p. 1119), the bladder is acontractile and areflexic (detrusor areflexia); sphincter tone usually persists and urinary retention is the rule (urinary incontinence generally does not occur except with overdistention). This requires catheter drainage (intermittent or indwelling) due to retention until the spinal shock resolves typically within 6 months[17]
      - after spinal shock subsides, most develop *detrusor hyperreflexia* → involuntary bladder contractions without sensation (automatic bladder), smooth sphincter synergy, but striated dyssynergy (involuntary contraction of the external sphincter during voiding which produces a functional outlet obstruction with poor emptying and high vesical pressures which is transmitted to the kidneys and may result in loss of renal function). Bladder fills and empties spontaneously due to reflexive voiding.[17] Bladder hypertrophy occurs due to contraction against a closed sphincter and bladder storage pressure increases. Patients have DO with DSD. Management goals: decrease bladder pressures and preserve renal function, usually with pharmaceuticals and intermittent catheterizations. The frequency of bladder drainage is determined by urodynamic pressures to ensure urine volumes consistent with safe storage pressures (see below).
   b) infrasacral lesions (lesion below the S2 spinal cord level): includes injury to conus medullaris, cauda equina or peripheral nerves (formerly referred to as lower motor neuron lesions). Etiologies: large HLD, trauma with compromise of spinal canal or peripheral nerve injuries (traumatic or iatrogenic with pelvic surgery). Detrusor areflexia usually ensues, and do not have voluntary or involuntary bladder contractions. Reduced urinary flow rate or retention results, and voluntary voiding may be lost. Overflow incontinence develops. Usually associated with loss of bulbocavernosus (BCR) and anal wink reflexes (preserved in suprasacral lesions, except when spinal shock is present (p. 1119)) and perineal sensory loss. NB: up to 20% of neurologically normal patients do not exhibit a BCR[18]
3. specific disease processes
   a) herniated lumbar disc (p. 1250): most consist initially of difficulty voiding, straining, or urinary retention. Later, irritative symptoms may develop
   b) spinal stenosis (lumbar or cervical): urologic symptoms vary, and depend on the spinal level (s) involved and the type of involvement (e.g., in cervical spinal stenosis, detrusor hyperactivity or underactivity may occur depending on whether the involvement of the micturition neural axis is compression of the inhibitory reticulospinal tracts or myelopathy involving the posterior funiculus)
   c) cauda equina syndrome (p. 1254): usually produces urinary retention, although sometimes incontinence may occur (some cases are overflow incontinence)

d) peripheral neuropathies: such as with diabetes, usually produce impaired detrusor activity
e) neurospinal dysraphism: most myelodysplastic patients have an areflexic bladder with an open bladder neck. The bladder usually fills until the resting residual fixed external sphincter pressure is exceeded and the leakage occurs
f) multiple sclerosis: 50–90% of patients develop voiding symptoms at some point. The demyelination primarily involves the posterior and lateral columns of the cervical spinal cord. Detrusor hyperreflexia is the most common urodynamic abnormality (in 50–99% of cases), with bladder areflexia being less common (5–20%). Patients have DO with DSD without upper tract injury or loss of compliance
g) tethered cord: urologic complaints are present on initial presentation 30–70% of the time. Most common urologic symptoms are urgency and incontinence. Urodynamic findings show DO with DSD.[19] Urinary dysfunction improves in more than half, but not all patients, after surgical correction[20]

Urinary retention

Etiologies of urinary retention:

1. bladder outlet obstruction (a brief differential diagnosis list is presented here)
   a) urethral stricture: retention tends to be progressive over time
   b) prostatic enlargement in males:
      - benign prostatic hypertrophy (BPH) & prostate cancer: retention tends to be progressive over time
      - acute prostatitis: onset of retention may be *sudden*
   c) women: bladder or vaginal prolapse which can produce a urethral kink
   d) obstructing thrombus from hematuria (clot retention)
   e) bladder calculi
   f) bladder or urethral foreign bodies
   g) urethral cancer: rare
2. detrusor areflexia or hypotonia
   a) spinal cord lesion
      - trauma
      - tumor
      - myelomeningocele
   b) cauda equina syndrome (p. 1254)
   c) medications: anticholinergics, narcotics
   d) diabetes mellitus (autonomic neuropathy)
   e) herpes zoster at the level of the sacral dorsal root ganglia[21 (p 967)]
3. postoperative urinary retention (POUR): occurs in ~ 4% of all surgeries, and 20–40% in neurosurgical patients after general anesthesia.[22,23] Felt to be secondary to combination of patient predisposition (eg BPH) along with anesthetic. Propofol, narcotics, benzodiazepines, inhaled anesthetics, and local intrathecal and epidural have all been shown to impact bladder contraction and coordination of micturition. POUR should be managed with CIC or indwelling catherization along with alpha blockers (see below) in men. Voiding trial may be done as soon as postoperative day 1 to avoid prolonged catheterization but keeping the Foley for 3–4 days has been shown to decrease need for replacement of the catheter.[23] POUR may persist > 1 week. Preoperative use of alpha blockers in at risk patients has shown protective against POUR in some studies, but not significant difference in other studies.[24] Urgent intervention is recommended to avoid long term sequela of bladder distention

Evaluation of bladder function

Urodynamics (UDS)

Usually combined with X-ray (cystometrogram [CMG]) or fluoro (videourodynamics). Measures intravesicular pressures during retrograde bladder filling through a urethral catheter, usually combined with sphincter electromyography. Assesses intravesical pressures during filling and voiding. Objectively assesses detrusor muscle at time of sensation to void. Most importantly, assesses bladder compliance, bladder storage pressures and risk for long term upper tract deterioration. Bladder pressures: < 40 cm H_20 is the cut off for safe storage pressures.[25] If bladder pressure > 40 cm H_20 during storage of urine, there is a high risk of progressive CKD. Routine UDS can help ensure safe management of a neurogenic bladder. UDS can also be used in the neurologically intact patient to determine if urinary retention is secondary to obstruction versus bladder areflexia.[26]

Voiding cystourethrogram and intravenous pelography (IVP)

Voiding cystourethrogram (VCUG) detects urethral pathology (diverticula, strictures…), abnormalities of bladder (diverticula, detrusor trabeculations associated with long-standing contractions against high resistance…), and vesical-ureteral reflux. VCUG can be performed at the time of UDS (video urodynamics).

3

Urologic follow-up

Routine follow-up is needed to ensure bladder pressures < 40 cm H_2O, and subsequently for periodic renal imaging and monitoring of serum creatinine.

Changes in voiding symptoms should trigger prompt reevaluation.

NB: patient with indwelling catheters (Foley, suprapubic tube…) or intermittent catheterization will have colonization of their urine. Treatment for positive urine cultures is only indicated when related symptoms develop or when undergoing instrumentation.

Pharmacologic treatment for bladder dysfunction

Muscarinic anticholinergics

Bladder contraction is produced by ACh-mediated stimulation of postganglionic parasympathetic muscarinic cholinergic receptors on bladder smooth muscle. Anticholinergics bind M2 and M3 choline receptors and prevent stimulation. This increases bladder capacity by 50 ml and decreases bladder storage pressures by 15 cm H_20.[27] They are a pillar in treating neurogenic bladders.

All are contraindicated in glaucoma as anticholinergics induce mydriasis. Overdosage results in the classic anticholinergic symptoms ("red as a beet, hot as a stove, dry as a rock, mad as a hatter"). Use is often limited by side effects including dry mouth, constipation, dry eyes, blurry vision, urinary retention & indigestion.

Anticholinergics may negatively impact cognition and memory.[28,29] Newer agents (tolterodine, darifenacin) have less impact on memory. Trospium, a quaternary amine, crosses the blood-brain barrier less readily than other anticholinergics and may have less negative impact.[29]

Drug info: Oxybutynin (Ditropan®)

Widely prescribed agent. Combines anticholinergic activity with independent musculotropic relaxant effect and local anesthetic activity. Immediate release (IR) produces the most side effects (including cognitive) in the class, which are better with extended release (ER).

℞ Adult IR: 5–30 mg divided TID. ER: 10–30 mg/d. Patch (Oxytrol) 3.7 mg q 3 d. ℞ Peds: not recommended for age < 5 years; usual dose is 5 mg BID (maximum 5 mg TID). **Supplied:** 5 mg tablets, 5 mg/5 ml syrup.

Drug info: Tolterodine (Detrol®)

Milder side effects than oxybutynin, but may also be less effective.[30] Side effects with IR > ER.

℞ IR: 2–8 mg PO divided BID. Can be lowered to 1 mg PO BID in some patients. ER: 2–8 mg qd. **Supplied:** 1 & 2 mg tablets. Detrol® LA 2 & 4 mg capsule

Drug info: Solifenacin (Vesicare®)

Most constipation in class.

℞5–10 mg qd. **Supplied:** ER & 10 mg tablets.

Drug info: Darifenacin (Enablex®)

℞5–10 mg po qd. **Supplied:** ER 7.5 & 15 mg tablets.

Drug info: Fesoterodine (Toviaz®)

℞4 mg PO qd, may increase up to 8 mg po qd PRN. **Supplied:** ER 4 & 8 mg tablets.

Drug info: Trospium (Sanctura®, Sanctura® XR)

℞IR: 20–60 mg po divided BID, ER: capsule 60 mg po qd. **Supplied:** IR: 20 mg tablets, ER: 60 mg capsule.

Alpha blockers

Alpha-adrenoreceptor antagonists block alpha-1 receptors on the bladder neck which results in smooth muscles relaxation and decreased bladder outlet resistance. This increases bladder compliance and decreases storage pressures with neurogenic bladders. Terazosin also decreases the frequency and severity of symptoms of autonomic dysreflexia.[27] Side effects include postural hypotension, rhinitis and retrograde ejaculation. Hypotension is more common in less selective alpha blockers and therefore dose escalation is required with terazosin and doxazosin.

Drug info: Tamsulosin (Flomax®)

A prostate alpha$_{1A}$ adrenoreceptor antagonist. Used to treat voiding difficulties resulting from outlet obstruction due to benign prostatic hypertrophy (BPH). Has some effectiveness in women via other mechanisms. Similar to terazosin (Hytrin®) and doxazosin (Cardura®), but has an advantage for acute relief because the dose of tamsulosin does not need to be gradually ramped up (it can be started at the therapeutic dose). It takes at least 5–7 days to work.

Side effects : very few. Rhinitis, retrograde or diminished ejaculation, or postural hypotension may occur.[31]

℞: 0.4 mg PO qd (usually given 30 minutes after the same meal each day). If there is no response by 2–4 weeks, a dose of 0.8 mg PO qd can be tried.[31]

Botulinum toxin (Botox™)

Botulinum toxin A (BTX-A) inhibits acetylcholine exocytosis from parasympathetic postganglionic nerves to the M2 and M3 receptors of the detrusor, inhibiting detrusor contraction. BTX-A (100–200u) is injected into the detrusor muscle during cystoscopy. It decreases overactivity, urgency, and storage pressures.[32] Efficacy lasts 3–12 months and repeat injections are required. Maximum Botox dose is 360 u per 90 day from all sources. Side effects include urinary tract infection and urinary retention. In patient not already managed with catherization, must be aware of the risk of urinary retention and need for temporary catheterization in 2–20% of patients.[32] This is usually self-limited to weeks or months as the BTX-A wears off.

Neuromodulation for bladder dysfunction

Permanent implantable neuromodulation (e.g., InterStim™ by Medtronic) is indicated for refractory urinary urgency, frequency, urge incontinence, non-obstructive urinary retention, and fecal incontinence. If a trial with a temporary lead placed adjacent to the sacral nerve via the S3 foramen produces > 50% reduction in symptoms, it is connected to an implantable pulse generator. Mechanism of action is poorly understood but may modulate the afferent signals of the micturition reflex.[33] Improvement in symptoms is seen in up to 70% of patients with complete resolution in incontinence around 39%.[34] Contraindications to implantation include failure to improve with trial and the likely need for repeated MRIs in the future (the device is MRI conditional for head only with ≤ 1.5 tesla MRI).

Bladder management after acute urinary retention

In situations where there is urinary retention (e.g., following cauda equina compression) with some prospect of return of function (e.g., following surgery for acute cauda equina compression) the following bladder management regimen may be employed:

- early bladder management is key to avoid bladder overdistention & permanent injury to the detrusor
- use of intermittent catheterization (if able to be performed), or indwelling catheter (Foley or suprapubic) to drain bladder with a goal of < 400 ml each time (or volumes lower than patient's safe bladder capacity if known)
- initiate alpha blockers (e.g., tamsulosin (Flomax®) (p. 97) 0.4 mg PO q d (see above)
- urology consultation for assistance with long-term follow-up & bladder management

3.2 Regional brain syndromes

This section serves to briefly describe typical syndromes associated with lesions in various areas of the brain. Unless otherwise noted, the described syndromes occur with *destructive* lesions.

3.2.1 Overview

1. frontal lobe
   a) unilateral injury:
      - may produce few clinical findings except with very large lesions
      - bilateral or large unilateral lesions: apathy, abulia
      - the frontal eye field (for contralateral gaze) is located in the posterior frontal lobe (Br. area 8, shown as the striped area in ▶ Fig. 1.1). Destructive lesions impair gaze to the contralateral side (patient looks *toward* the side of the lesion), whereas irritative lesions (i.e., seizures) cause the center to activate, producing contralateral gaze (patient looks *away* from the side of the lesion). See also **Extraocular muscle (EOM) system** (p. 596) for more details.

   b) bilateral injury: may produce apathy, abulia
   c) olfactory groove region: may produce Foster Kennedy syndrome (p. 100)
   d) prefrontal lobes control "executive function": planning, prioritizing, organizing thoughts, suppressing impulses, understanding the consequences of decisions
2. parietal lobe: major features (see below for details)
   a) either side: cortical sensory syndrome, sensory extinction, contralateral homonymous hemianopia, contralateral neglect
   b) dominant parietal lobe lesion (left in most): language disorders (aphasias), Gerstmann syndrome (p. 99), bilateral astereognosis
   c) non-dominant parietal lobe lesions: topographic memory loss, anosognosia and dressing apraxia
3. occipital lobe: homonymous hemianopsia
4. cerebellum
   a) lesions of the cerebellar hemisphere cause ataxia in the *ipsilateral* limbs
   b) lesions of the cerebellar vermis cause truncal ataxia
5. brainstem: usually produces a mixture of cranial nerve deficits and long tract findings (see below for some specific brainstem syndromes)
6. pineal region
   a) Parinaud's syndrome (p. 101)

3.2.2 Parietal lobe syndromes

See reference.[35 (p 308–12)]

Parietal lobe anatomy

The parietal lobe is located behind the central sulcus, above the Sylvian fissure, merging posteriorly into the occipital lobe (the border on the medial surface of the brain is defined by a line connecting the parieto-occipital sulcus to the pre-occipital notch).

Parietal lobe neurophysiology

- either side: anterior parietal cortex organizes tactile precepts (probably contralateral) and integrates with visual and auditory sensation to build awareness of body and its spatial relations
- dominant side (on left in 97% of adults): understanding language, includes "cross-modal matching" (auditory-visual, visual-tactile, etc.). Dysphasia present with dominant lobe lesions often impedes assessment
- non-dominant side (right in most): integrates visual and proprioceptive sensation to allow manipulation of body and objects, and for certain constructional activities

Clinical syndromes of parietal lobe disease

Overview

1. *unilateral* parietal lobe disease (dominant or non-dominant):
   a) cortical sensory syndrome (see below) and sensory extinction (neglecting 1 of 2 simultaneously presented stimuli). Large lesion → hemianesthesia
   b) congenital injury → mild hemiparesis & contralateral muscle atrophy
   c) homonymous hemianopia or visual inattentiveness
   d) occasionally: anosognosia
   e) neglect of contralateral half of body and visual space (more common with right side lesions)
   f) abolition of *optokinetic nystagmus* to one side
2. additional effects of dominant parietal lobe lesion (left in most people):
   a) language disorders (aphasias)
   b) speech-related or verbally mediated functions, e.g., cross-modal matching (e.g., patient understands spoken words and can read, but cannot understand sentences with elements of relationships)
   c) Gerstmann syndrome, named for Josef Gerstmann (1887-1969). Localizes to the angular and supramarginal gyrus (Brodmann area 39 & 40 respectively) of the dominant hemispehre. Classically:
      - agraphia without alexia (patients cannot write but can still read)
      - left-right confusion
      - digit agnosia: inability to identify finger by name
      - acalculia (or dyscalculia): difficulty with math
   d) tactile agnosia (bilateral astereognosis)
   e) bilateral ideomotor apraxia (inability to carry out verbal commands for activities that can otherwise be performed spontaneously with ease)
3. additional effects of non-dominant parietal lobe lesions (usually right):
   a) topographic memory loss
   b) anosognosia and dressing apraxia

Cortical sensory syndrome

Lesion of postcentral gyrus, especially area that maps to hand.

- sensory deficits:
  a) loss of position sense and of passive movement sense
  b) inability to localize tactile, thermal, and noxious stimuli
  c) astereognosis (inability to judge object size, shape, and identity by feel)
  d) agraphesthesia (cannot interpret numbers written on hand)
  e) loss of two point discrimination
- preserved sensations: pain, touch, pressure, vibration, temperature
- other features
  a) easy fatigability of sensory perceptions
  b) difficulty distinguishing simultaneous stimulations
  c) prolongation of superficial pain with hyperpathia
  d) touch hallucinations

Anton-Babinski syndrome

A unilateral asomatognosia. May seem more common with non-dominant (usually right) parietal lesions because it may be obscured by the aphasia that occurs with dominant (left) sided lesions.