CHAPTER e47 Special Issues in Inpatient Neurologic Consultation

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Inpatient neurologic consultations usually involve questions about specific disease processes or prognostication after various cerebral injuries. Common reasons for neurologic consultation include stroke (Chap. 370), seizures (Chap. 369), altered mental status (Chap. 25), headache (Chap. 14), and management of coma and other neurocritical care conditions (Chaps. 274 and 275). This chapter focuses on additional common reasons for consultation that are not addressed elsewhere in the text.

CONSULTATIONS REGARDING CENTRAL NERVOUS SYSTEM DYSFUNCTION

HYPERPERFUSION STATES

A group of neurologic disorders shares the common feature of hyperperfusion playing a key role in pathogenesis. These seemingly diverse syndromes include hypertensive encephalopathy, eclampsia, postcarotid endarterectomy syndrome, and toxicity from calcineurin-inhibitor medications. Modern imaging techniques and experimental models suggest that vasogenic edema is usually the primary process leading to neurologic dysfunction; therefore, prompt recognition and management of this condition should allow for clinical recovery if superimposed hemorrhage or infarction has not occurred.

The brain's autoregulatory capability successfully maintains a fairly stable cerebral blood flow in adults despite alterations in systemic mean arterial pressure (MAP) ranging from 50–150 mm Hg. In patients with chronic hypertension, this cerebral autoregulation curve is shifted, resulting in autoregulation working over a much higher range of pressures (e.g., 70–175 mm Hg). In these hypertensive patients, cerebral blood flow is kept steady at higher MAP, but a rapid lowering of pressure can more easily lead to ischemia on the lower end of the autoregulatory curve. This autoregulatory phenomenon is achieved through both myogenic and neurogenic influences causing small arterioles to contract and dilate. When the systemic blood pressure exceeds the limits of this mechanism, breakthrough of autoregulation occurs, resulting in hyperperfusion via increased cerebral blood flow, capillary leakage into the interstitium, and resulting edema. The predilection of all of the hyperperfusion disorders to affect the posterior rather than anterior portions of the brain may be due to a lower threshold for autoregulatory breakthrough in the posterior circulation.

While elevated or relatively elevated blood pressure is common in many of these disorders, some hyperperfusion states such as calcineurin-inhibitor toxicity occur with no apparent pressure rise. In these cases, vasogenic edema is likely due primarily to dysfunction of the capillary endothelium itself, leading to breakdown of the blood-brain barrier. It is useful to separate disorders of hyperperfusion into those caused primarily by increased pressure and those due mostly to endothelial dysfunction from a toxic or autoimmune etiology (Table e47-1). In reality, both of these pathophysiologic processes are likely playing some role in each of these disorders.

TABLE e47-1 Some Common Etiologies of Hyperperfusion Syndrome

Disorders in which increased capillary pressure dominates the pathophysiology
Hypertensive encephalopathy, including secondary causes such as renovascular hypertension, pheochromocytoma, cocaine use, etc.
Postcarotid endarterectomy syndrome
Preeclampsia/eclampsia
High-altitude cerebral edema
Disorders in which endothelial dysfunction dominates the pathophysiology
Calcineurin-inhibitor toxicity
Chemotherapeutic agent toxicity (e.g., cytarabine, azathioprine, 5-fluorouracil, cisplatin, methotrexate)
Glucocorticoids
Erythropoietin
HELLP syndrome (*h*emolysis, *e*levated /iver enzyme levels, /ow *p*latelet count)
Thrombotic thrombocytopenic purpura (TTP)
Hemolytic uremic syndrome (HUS)
Systemic lupus erythematosus (SLE)
Granulomatosis with polyangiitis (Wegener's)

The clinical presentation of the hyperperfusion syndromes is similar with prominent headaches, seizures, or focal deficits. Headaches have no specific characteristics, range from mild to severe, and may be accompanied by alterations in consciousness ranging from confusion to coma. Seizures may be present, and these can be of multiple types depending on the severity and location of the edema. Nonconvulsive seizures have been described in hyperperfusion states; therefore, a low threshold for obtaining an electroencephalogram (EEG) in these patients should be maintained. The typical focal deficit in hyperperfusion states is cortical visual loss, given the tendency of the process to involve the occipital lobes. However, any focal deficit can occur depending on the area affected, as evidenced by patients who, after carotid endarterectomy, exhibit neurologic dysfunction in the ipsilateral newly reperfused hemisphere. In conditions where increased cerebral blood flow plays a role, examination of the inpatient vital signs record will usually reveal a systemic blood pressure that is increased above baseline. It appears as if the rapidity of rise rather than the absolute value of pressure is the most important risk factor.

The diagnosis in all of these conditions is clinical. The symptoms of these disorders are common and nonspecific, so a long differential diagnosis should be entertained, including consideration of other causes of confusion, focal deficits, headache, and seizures. MRI has improved the ability of clinicians to diagnose hyperperfusion syndromes, although cases have been reported with normal imaging. Patients classically exhibit the high T2 signal of edema primarily in the posterior occipital lobes, not respecting any single vascular

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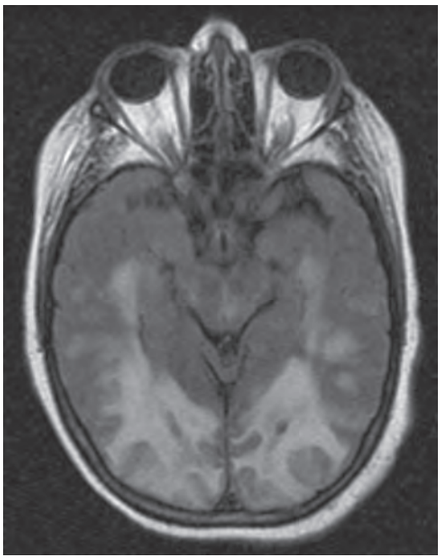

Figure e47-1 Axial fluid-attenuated inversion recovery (FLAIR) MRI of the brain in a patient taking cyclosporine after liver transplantation, who presented with seizures, headache, and cortical blindness. Increased signal is seen bilaterally in the occipital lobes predominantly involving the white matter, consistent with a hyperperfusion state secondary to calcineurin-inhibitor exposure.

territory (Fig. e47-1). Diffusion-weighted images are typically normal, emphasizing the vasogenic rather than cytotoxic nature of this edema. Imaging with CT is less sensitive but may show a pattern of patchy hypodensity in the involved territory. Previously this classic radiographic appearance had been termed *reversible posterior leukoencephalopathy* (RPLE). However, this term has fallen out of favor because none of its elements are completely accurate. The radiographic and clinical changes are not always reversible; the territory involved is not uniquely posterior; and gray matter may be affected as well, rather than purely white matter as the word "leukoencephalopathy" intimates. Other ancillary studies such as cerebrospinal fluid (CSF) analysis often yield nonspecific results. It should be noted that many of the substances that have been implicated, such as cyclosporine, can cause this syndrome even at low doses or after years of treatment. Therefore, normal serum levels of these medications do not exclude them as inciting agents.

In cases of hyperperfusion syndromes, treatment should commence urgently once the diagnosis is considered. Hypertension plays a key role commonly, and judicious lowering of the blood pressure with IV agents such as labetalol or nicardipine is advised along with continuous cardiac and blood pressure monitoring, often through an arterial line. It is reasonable to lower mean arterial pressure by ~20% initially, as further lowering of the pressure may cause secondary ischemia as pressure drops below the lower range of the patient's autoregulatory capability. In cases where there is an identified cause of the syndrome, these etiologies should be treated promptly, including discontinuation of offending substances such as calcineurin inhibitors in toxic processes, treatment of immune-mediated disorders such as thrombotic thrombocytopenic purpura (TTP), and prompt delivery of the fetus in eclampsia. Seizures must be identified and controlled, often necessitating continuous EEG monitoring. Anticonvulsants are effective, but in the special case of eclampsia, there is good evidence to support the use of magnesium sulfate for seizure control.

POST-CARDIAC BYPASS BRAIN INJURY

Central nervous system (CNS) injuries following open heart or coronary artery bypass grafting (CABG) surgery are common and include acute encephalopathy, stroke, and a chronic syndrome of cognitive impairment, which is now increasingly recognized. Hypoperfusion and embolic disease are frequently involved in the pathogenesis of these syndromes, although multiple mechanisms may be involved in these critically ill patients who are at risk for various metabolic and polypharmaceutical complications.

The frequency of hypoxic injury secondary to inadequate blood flow intraoperatively has been markedly decreased by the use of modern surgical and anesthetic techniques. Despite these advances, some patients still experience neurologic complications from cerebral hypoperfusion or may suffer focal ischemia from tight carotid or focal intracranial stenoses in the setting of regional hypoperfusion. Postoperative infarcts in the border zones between vascular territories commonly are blamed on systemic hypotension although some have suggested that these infarcts can also result from embolic disease (Fig. e47-2).

Embolic disease is likely the predominant mechanism of cerebral injury during cardiac surgery as evidenced by diffusion-weighted MRI and intraoperative transcranial Doppler studies. It should be noted that some of the emboli that are found histologically in these patients are too small to be detected by standard imaging sequences; therefore, a negative MRI after surgery does not exclude the diagnosis of emboli-related complications. Thrombus in the heart itself as well as atheromas in the aortic arch can become dislodged during cardiac surgeries, releasing a shower of particulate matter into the cerebral circulation. Cross-clamping of the aorta, manipulation of the heart, extracorporeal circulation techniques ("bypass"), arrhythmias such as atrial fibrillation, and introduction of air through suctioning have all been implicated as potential sources of emboli. Histologic studies indicate that literally millions of tiny emboli may be released, even using modern surgical techniques.

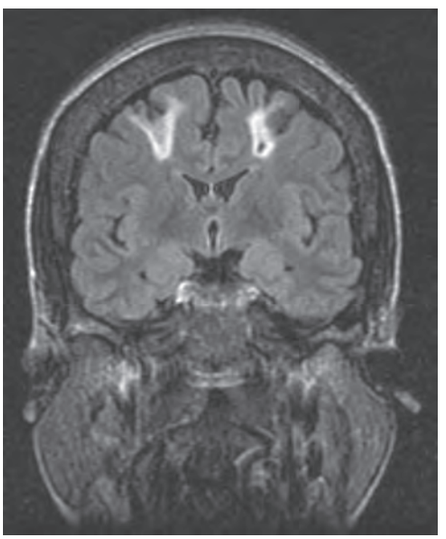

Figure e47-2 Coronal fluid-attenuated inversion recovery (FLAIR) MRI of the brain in a patient presenting with altered mental status after an episode of hypotension during coronary artery bypass grafting (CABG). Increased signal is seen in the border zones bilaterally between the middle cerebral artery and anterior cerebral artery territories. Diffusion-weighted MRI sequences demonstrated restricted diffusion in these same locations, suggesting acute infarction.

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This shower of microemboli results in a number of clinical syndromes. Occasionally, a single large embolus leads to an isolated large-vessel stroke that presents with obvious clinical focal deficits. More commonly, the emboli released are multiple and smaller. When there is a high burden of these small emboli, an acute encephalopathy can occur postoperatively, presenting as either a hyperactive or hypoactive confusional state, the latter of which is frequently and incorrectly ascribed to depression. When the burden of microemboli is lower, no acute syndrome is recognized, but the patient may suffer a chronic cognitive deficit. Cardiac surgery can be viewed, like delirium, as a "stress test for the brain." Some patients with a low cerebral reserve due to underlying cerebrovascular disease or an early neurodegenerative process will develop a chronic, cognitive deficit, whereas others with higher reserves may remain asymptomatic despite a similar dose of microemboli. In this manner, cardiac surgery may serve to unmask the early manifestations of disorders such as vascular dementia and Alzheimer's disease.

Since modern techniques have successfully minimized hypoperfusion complications during these surgeries, much attention is now focused on reducing this inevitable shower of microemboli. Off-pump CABG surgeries have the advantages of reducing length of stay and perioperative complications; however, some recent data suggests that off-pump CABG does not preserve cognitive function compared with on-pump CABG. Filters placed in the aortic arch may have some promise in capturing these emboli, although convincing evidence is currently lacking. Development of successful endovascular operative approaches may provide a reasonable alternative to conventional CABG procedures, especially for patients at high risk of developing cognitive dysfunction after surgery due to advanced age, previous stroke, or severe atheromatous disease of the carotid arteries or aortic arch.

POST-SOLID ORGAN TRANSPLANT BRAIN INJURY

Patients who have undergone solid organ transplantation are at risk for neurologic injury in the postoperative period and for the months to years thereafter. Neurologic consultants should view these patients as a special population at risk for both unique neurologic complications as well as for the usual disorders found in any critically ill inpatient.

Immunosuppressive medications are administered in high doses to patients after solid organ transplant, and many of these compounds have well-described neurologic complications. In patients with headache, seizures, or focal neurologic deficits taking calcineurin inhibitors, the diagnosis of hyperperfusion syndrome should be considered, as discussed above. This neurotoxicity occurs mainly with cyclosporine and tacrolimus and can present even in the setting of normal serum drug levels. Treatment primarily involves lowering the drug dosage or discontinuing the drug. A related newer agent, sirolimus, has very few recorded cases of neurotoxicity and may be a reasonable alternative for some patients. Other examples of immunosuppressive medications and their neurologic complications include OKT3-associated akinetic mutism and the leukoencephalopathy seen with methotrexate, especially when it is administered intrathecally or with concurrent radiotherapy. In any solid organ transplant patient with neurologic complaints, a careful examination of the medication list is required to search for these possible drug effects.

Cerebrovascular complications of solid organ transplant are often first recognized in the immediate postoperative period. Border zone territory infarctions can occur, especially in the setting of systemic hypotension during cardiac transplant surgery. Embolic infarctions classically complicate cardiac transplantation, but all solid organ transplant procedures place patients at risk for systemic emboli. When cerebral embolization accompanies renal or liver transplantation surgery, a careful search for right-to-left shunting should include evaluation of the heart with agitated saline echocardiography, as well as looking for intrapulmonary shunting. Renal and some cardiac transplant patients often have advanced atherosclerosis, providing yet another mechanism for stroke. Imaging with CT or MRI with diffusion is advised when cerebrovascular complications are suspected to confirm the diagnosis and to exclude intracerebral hemorrhage, which most often occurs in the setting of coagulopathy secondary to liver failure or after cardiac bypass procedures.

Given that patients with solid organ transplants are chronically immunosuppressed, infections are a common concern (Chap. 132). In any transplant patient with new CNS signs or symptoms such as seizure, confusion, or focal deficit, the diagnosis of a central nervous system infection should be considered and evaluated through imaging (usually MRI) and possibly lumbar puncture. The most common pathogens responsible for CNS infections in these patients vary based on time since transplant. In the first month posttransplant, common pathogens include the usual bacterial organisms associated with surgical procedures and indwelling catheters. Starting in the second month posttransplant, opportunistic infections of the CNS become more common, including *Nocardia* and *Toxoplasma* species as well as fungal infections such as aspergillosis. Viral infections that can affect the brain of the immunosuppressed patient, such as herpes simplex virus, cytomegalovirus, and varicella, also become more common after the first month posttransplant. After 6 months posttransplant, immunosuppressed patients still remain at risk for these opportunistic bacterial, fungal, and viral infections but can also suffer late CNS infectious complications such as progressive multifocal leukoencephalopathy (PML) associated with JC virus and Epstein-Barr virus–driven clonal expansions of B cells resulting in CNS lymphoma.

COMMON NEUROLOGIC COMPLICATIONS OF ELECTROLYTE DISTURBANCES

A wide variety of neurologic conditions can result from abnormalities in serum electrolytes, and consideration of electrolyte disturbances should be part of any inpatient neurologic consultation. **A complete general discussion of fluid and electrolyte imbalance and homeostasis can be found in Chap. 45.**

HYPERNATREMIA AND HYPEROSMOLALITY

The normal range of serum osmolality is around 275–295 mOsm/kg, but neurologic manifestations are usually seen only at levels >325 mOsm/kg. Hyperosmolality is usually due to hypernatremia, hyperglycemia, azotemia, or the addition of extrinsic osmoles such as mannitol, which is commonly used in critically ill neurologic patients. Hyperosmolality itself can lead to a generalized encephalopathy that is nonspecific and without focal findings; however, an underlying lesion such as a mass can become symptomatic under the metabolic stress of a hyperosmolar state, producing focal signs. Some patients with hyperosmolality from severe hyperglycemia can present, for unclear reasons, with generalized seizures or unilateral movement disorders, which usually respond to lowering of the serum glucose. The treatment of all forms of hyperosmolality involves calculation of apparent water losses and slow replacement so that the serum sodium declines no faster than 2 mmol/L (2 meq/L) per hour.

Hypernatremia leads to the loss of intracellular water, leading to cell shrinkage. In the cells of the brain, solutes such as glutamine and urea are generated under these conditions in order to minimize this shrinkage. Despite this corrective mechanism, when hypernatremia is severe [serum sodium >160 mmol/L (>160 meq/L)] or occurs rapidly, cellular metabolic processes fail and encephalopathy

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will result. There are many etiologies of hypernatremia including, most commonly, renal and extrarenal losses of water. Causes of neurologic relevance include central diabetes insipidus, where hyperosmolality is accompanied by submaximal urinary concentration due to inadequate release of arginine vasopressin (AVP) from the posterior pituitary, resulting often from pituitary injury in the setting of surgery, hemorrhage, infiltrative processes, or cerebral herniation.

HYPONATREMIA

Hyponatremia is commonly defined as a serum sodium <135 mmol/L (<135 meq/L). Neurologic symptoms occur at different levels of low sodium, depending not only on the absolute value but also on the rate of fall. In patients with hyponatremia that develops over hours, life-threatening seizures and cerebral edema may occur at values as high as 125 mmol/L. In contrast, some patients with more chronic hyponatremia that has slowly developed over months to years may be asymptomatic even with serum levels <110 mmol/L. Correction of hyponatremia, especially when chronic, must take place slowly in order to avoid additional neurologic complications. Cells in the brain swell in hypotonic hyponatremic states but may compensate over time by excreting solute into the extracellular space, leading to restoration of cell volume when water follows the solute out of the cells. If treatment of hyponatremia results in a rapid rise in serum sodium, cells in the brain may quickly shrink, leading to osmotic demyelination, a process that previously was thought to be limited exclusively to the brainstem (central pontine myelinolysis; see Fig. 275-6), but now has been described elsewhere in the CNS.

Treatment of hyponatremia is dependent on the cause. Hypertonic hyponatremia treatment focuses on the underlying condition, such as hyperglycemia. Isovolemic hyponatremia [syndrome of inappropriate antidiuretic hormone (SIADH)] is managed with water restriction or administration of AVP antagonists. The management of choice for patients with hypervolemic hypotonic hyponatremia is free-water restriction and treatment of the underlying edematous disorder, such as nephrotic syndrome or congestive heart failure. Finally, in hypovolemic hypotonic hyponatremia, volume is replaced with isotonic saline while underlying conditions of the kidneys, adrenals, and gastrointestinal tract are addressed.

One neurologic cause of hypovolemic hypotonic hyponatremia is the cerebral salt-wasting syndrome that accompanies subarachnoid hemorrhage and, less commonly, other cerebral processes such as meningitis or stroke. In these cases, the degree of renal sodium excretion can be remarkable, and large amounts of saline, hypertonic saline, or oral sodium may need to be given in a judicious fashion in order to avoid complications from cerebral edema.

HYPOKALEMIA

Hypokalemia, defined as a serum potassium level < 3.5 mmol/L (<3.5 meq/L), occurs either because of excessive potassium losses (from the kidneys or gut) or due to an abnormal potassium distribution between the intracellular and extracellular spaces. At very low levels (<1.5 mmol/L), hypokalemia may be life threatening due to the risk of cardiac arrhythmia and may present neurologically with severe muscle weakness and paralysis. Hypokalemic periodic paralysis is a rare disorder caused by excessive intracellular potassium uptake in the setting of a calcium or sodium channel mutation. Treatment of hypokalemia is dependent on the etiology but usually includes replacement of potassium through oral or IV routes as well as correcting the cause of potassium balance problems (e.g., eliminating β_2-adrenergic agonist medications).

HYPERKALEMIA

Hyperkalemia is defined as a serum potassium level >5.5 mmol/L (>5.5 meq/L) and can neurologically present as muscle weakness with or without paresthesias. Hyperkalemia becomes life threatening when it produces electrocardiographic abnormalities such as peaked T waves or a widened QRS complex. In these cases, prompt treatment is essential and consists of strategies that protect the heart against arrhythmias (calcium gluconate administration); promote potassium redistribution into cells (with glucose, insulin, and β_2-agonist medications); and increase potassium removal (through sodium polystyrene sulfonate, loop diuretics, or hemodialysis).

CALCIUM DISTURBANCES

Hypercalcemia usually occurs in the setting of either hyperparathyroidism or systemic malignancy. Neurologic manifestations include encephalopathy as well as muscle weakness due to reduced neuromuscular excitability. Seizures can occur but are more common in states of low calcium.

Hypocalcemia in adults often follows surgical treatment of the thyroid or parathyroid. Seizures and altered mental status dominate the neurologic picture and usually resolve with calcium repletion. Tetany is due to spontaneous, repetitive action potentials in peripheral nerves and remains the classic sign of symptomatic hypocalcemia.

MAGNESIUM DISTURBANCES

Disorders of magnesium are difficult to correlate with serum levels because a very small amount of total-body magnesium is located in the extracellular space. Hypomagnesemia presents neurologically with seizures, tremor, and myoclonus. When intractable seizures occur in the setting of hypomagnesemia, only administration of magnesium will lead to resolution. High levels of magnesium, in contrast, lead to CNS depression. Hypermagnesemia usually only occurs in the setting of renal failure or magnesium administration and can lead to confusion and muscular paralysis when severe.

CONSULTATIONS REGARDING PERIPHERAL NERVOUS SYSTEM DYSFUNCTION

ENTRAPMENT NEUROPATHIES

Polyneuropathy is a common cause of outpatient neurologic consultation (Chap. 384). In the inpatient setting, however, mononeuropathies are more frequent, especially the entrapment neuropathies that complicate many surgical procedures and medical conditions. Median neuropathy at the wrist (carpal tunnel syndrome) is the most frequent entrapment neuropathy by far, but it is rarely a cause for inpatient consultation. Mechanisms for perioperative mononeuropathy include traction, compression, and ischemia of the nerve. Imaging with MR neurography may allow these causes to be distinguished definitively. In all cases of mononeuropathy, the diagnosis can be made through the clinical examination and then confirmed with electrodiagnostic studies in the subacute period, if necessary. Treatment consists mainly of avoidance of repetitive trauma but may also include surgical approaches to relieve pressure on the nerve.

RADIAL NEUROPATHY

Radial nerve injury classically presents with weakness of extension of the wrist and fingers ("wrist drop") with or without more proximal weakness of extensor muscles of the upper extremity, depending on the site of injury. Sensory loss is in the distribution of the radial nerve, which includes the dorsum of the hand (Fig. e47-3*A*). Compression at the level of the axilla, e.g., resulting from use of crutches, includes weakness of the triceps, brachioradialis, and supinator muscles in

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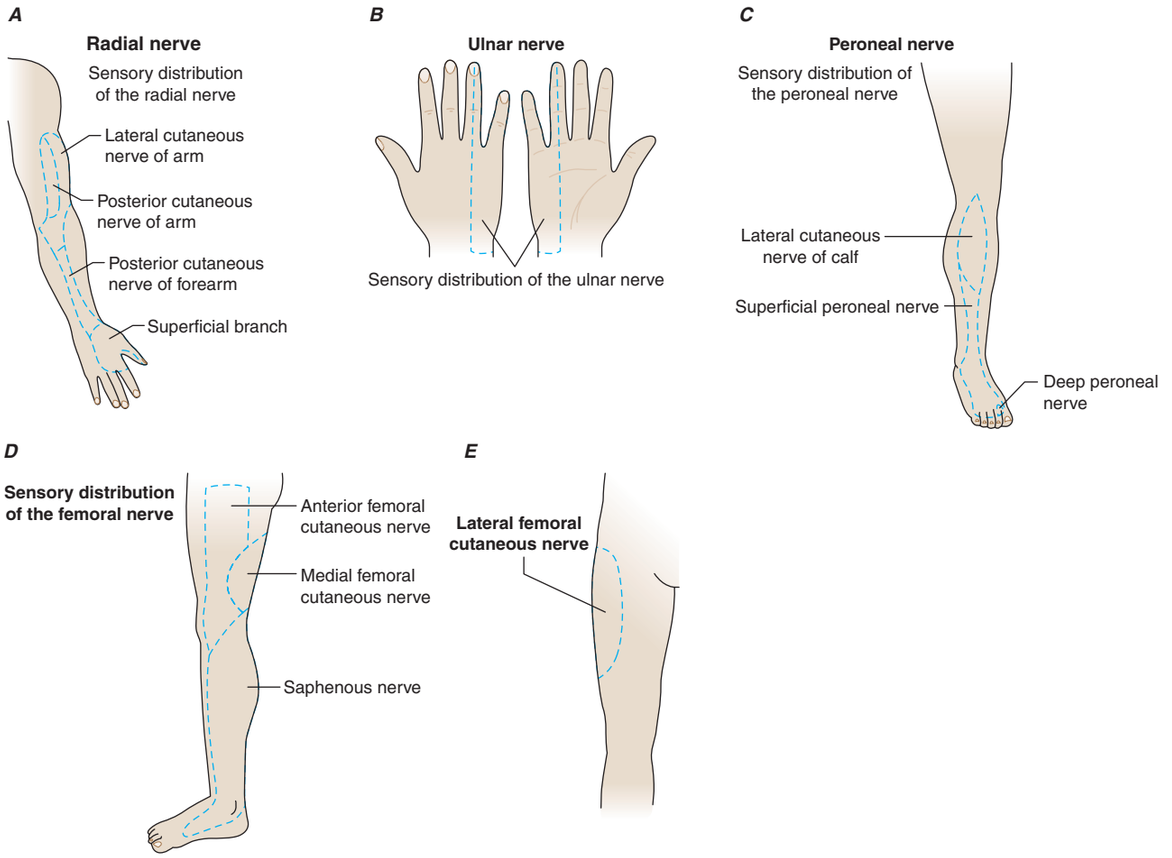

Figure e47-3 **Sensory distribution of peripheral nerves commonly affected by entrapment neuropathies.** ***A.*** Radial nerve. ***B.*** Ulnar nerve. ***C.*** Peroneal nerve. ***D.*** Femoral nerve. ***E.*** Lateral femoral cutaneous nerve.

addition to wrist drop. A more common site of compression occurs in the spiral groove of the upper arm in the setting of a humerus fracture or from sleeping with the arm draped over a bench or chair ("Saturday night palsy"). Sparing of the triceps is the rule when the nerve is injured in this location. Because extensors of the upper extremity are injured preferentially in radial nerve injury, these lesions may be mistaken for the pyramidal distribution of weakness that accompanies upper motor neuron lesions from brain or spinal cord processes.

ULNAR NEUROPATHY

Compression of the ulnar nerve is the second most common entrapment neuropathy after carpal tunnel syndrome. The most frequent site of compression is at the elbow where the nerve passes superficially in the ulnar groove. Symptoms usually begin with tingling in the ulnar distribution, including the fourth and fifth digits of the hand (Fig. e47-3*B*). Sensory symptoms may be worsened by elbow flexion due to increased pressure on the nerve, hence the tendency of patients to complain of increasing paresthesias at night when the arm is flexed at the elbow during sleep. Motor dysfunction can be disabling and involves most of the intrinsic hand muscles, limiting dexterity and strength of grasp and pinch. Etiologies of ulnar entrapment include trauma to the nerve (hitting the "funny bone"), malpositioning during anesthesia for surgical procedures, and chronic arthritis of the elbow. When a perioperative ulnar nerve injury is considered, stretch injury or trauma to the lower trunk of the brachial plexus should be entertained as well since its symptoms can mimic those of an ulnar neuropathy. If the clinical examination is equivocal, electrodiagnostic studies can definitively distinguish between plexus and ulnar nerve lesions a few weeks after the injury. Conservative methods of treatment are often the first step, but a variety of surgical approaches may be effective, including anterior ulnar nerve transposition and release of the flexor carpi ulnaris aponeurosis.

PERONEAL NEUROPATHY

The peroneal nerve winds around the head of the fibula in the leg below the lateral aspect of the knee, and its superficial location at this site makes it vulnerable to trauma. Patients present with weakness of foot dorsiflexion ("foot drop") as well as with weakness in eversion but not inversion at the ankle. Sparing of inversion, which is a function of muscles innervated by the tibial nerve, helps to distinguish peroneal neuropathies from L5 radiculopathies. Sensory loss involves the lateral aspect of the leg as well as the dorsum of the foot (Fig. e47-3*C*). Fractures of the fibular head may be responsible for peroneal neuropathies, but in the perioperative setting poorly applied braces exerting pressure on the nerve while the patient is unconscious are more often responsible. Tight-fitting stockings or casts of the upper leg can also cause a peroneal neuropathy, and thin individuals and those with recent weight loss are at increased risk.

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PROXIMAL FEMORAL NEUROPATHY

Lesions of the proximal femoral nerve are relatively uncommon but may present dramatically with weakness of hip flexion, quadriceps atrophy, weakness of knee extension (often manifesting with leg-buckling falls), and an absent patellar reflex. Adduction of the thigh is spared as these muscles are supplied by the obturator nerve, thereby distinguishing a femoral neuropathy from a more proximal lumbosacral plexus lesion. The sensory loss found is in the distribution of the femoral nerve sensory branches on the anterior part of the thigh (Fig. e47-3*D*). Compressive lesions from retroperitoneal hematomas or masses are common, and a CT of the pelvis should be obtained in all cases of femoral neuropathy to exclude these conditions. Bleeding into the pelvis resulting in hematoma can occur spontaneously, following trauma, or after intrapelvic surgeries such as renal transplantation. In intoxicated or comatose patients, stretch injuries to the femoral nerve are seen following prolonged, extreme hip flexion or extension. Rarely, attempts at femoral vein or arterial puncture can be complicated by injury to this nerve.

LATERAL FEMORAL CUTANEOUS NERVE

The symptoms of lateral femoral cutaneous nerve entrapment, commonly known as "meralgia paresthetica," include sensory loss, pain, and dysesthesia in part of the area supplied by the nerve (Fig. e47-3*E*). There is no motor component to the nerve, and therefore weakness is not a part of this syndrome. Symptoms often are worsened by standing or walking. Compression of the nerve occurs where it enters the leg near the inguinal ligament, usually in the setting of tight-fitting belts, pants, corsets, or recent weight gain, including that of pregnancy. The differential diagnosis of these symptoms includes hip problems such as trochanteric bursitis.

OBSTETRIC NEUROPATHIES

Pregnancy and delivery place women at special risk for a variety of nerve injuries. Radiculopathy due to a herniated lumbar disc is not common during pregnancy, but compressive injuries of the lumbosacral plexus do occur secondary to either the fetal head passing through the pelvis or the use of forceps during delivery. These plexus injuries are more frequent with cephalopelvic disproportion and often present with a painless unilateral foot drop which must be distinguished from a peroneal neuropathy caused by pressure on the nerve while in lithotomy position during delivery. Other compressive mononeuropathies of pregnancy include meralgia paresthetica, carpal tunnel syndrome, femoral neuropathy when the thigh is abducted severely in an effort to facilitate delivery of the fetal shoulder, and obturator neuropathy during lithotomy positioning. The latter presents with medial thigh pain that may be accompanied by weakness of thigh adduction. There is also a clear association between pregnancy and an increased frequency of idiopathic facial palsy (Bell's palsy).

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