

# Bell's Palsy

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## ABSTRACT

**Purpose of Review:** Bell's palsy is a common outpatient problem, and while the diagnosis is usually straightforward, a number of diagnostic pitfalls can occur, and a lengthy differential diagnosis exists. Recognition and management of Bell's palsy relies on knowledge of the anatomy and function of the various motor and nonmotor components of the facial nerve. Avoiding diagnostic pitfalls relies on recognizing red flags or features atypical for Bell's palsy, suggesting an alternative cause of peripheral facial palsy.

**Recent Findings:** The first American Academy of Neurology (AAN) evidence-based review on the treatment of Bell's palsy in 2001 concluded that corticosteroids were probably effective and that the antiviral acyclovir was possibly effective in increasing the likelihood of a complete recovery from Bell's palsy. Subsequent studies led to a revision of these recommendations in the 2012 evidence-based review, concluding that corticosteroids, when used shortly after the onset of Bell's palsy, were "highly likely" to increase the probability of recovery of facial weakness and should be offered; the addition of an antiviral to steroids may increase the likelihood of recovery but, if so, only by a very modest effect.

**Summary:** Bell's palsy is characterized by the spontaneous acute onset of unilateral peripheral facial paresis or palsy in isolation, meaning that no features from the history, neurologic examination, or head and neck examination suggest a specific or alternative cause. In this setting, no further testing is necessary. Even without treatment, the outcome of Bell's palsy is favorable, but treatment with corticosteroids significantly increases the likelihood of improvement.

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## INTRODUCTION

Bell's palsy is a common outpatient problem, and while the diagnosis is usually straightforward, a long and broad differential diagnosis exists for peripheral facial nerve palsy, and approximately one-third of cases are due to another cause. Bell's palsy is characterized by the acute spontaneous onset (72 hours or fewer) of unilateral peripheral facial paresis or palsy in isolation (no other neurologic or systemic signs), for which no specific etiology is uncovered, and in almost all cases, shows improvement within several months.<sup>1</sup> If a specific cause is found, such as Lyme disease or sarcoidosis, it should not be referred to as Bell's palsy.

## HISTORY OF BELL'S PALSY

Sir Charles Bell (1774 to 1842) is associated with idiopathic peripheral facial palsy, not because he was the first to observe or report this finding, since depictions of facial palsy can be traced to ancient art and texts (Figure 5-1),<sup>2</sup> but instead because Bell recognized that peripheral facial palsy resulted from involvement of the seventh cranial nerve (which he referred to as the respiratory nerve). As he demonstrated in a series of clinical and experimental observations, the seventh cranial nerve controlled the muscles of facial expression, as stated by Bell:

*On cutting the respiratory nerve on one side of the face of a*

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Dr Reich discusses the use of acyclovir and valacyclovir for the treatment of Bell's palsy.

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**KEY POINTS**

- Bell's palsy is characterized by the spontaneous acute (72 hours or fewer) onset of a unilateral peripheral facial nerve palsy without any accompanying signs, with neither the history nor examination suggesting an alternative diagnosis.
- Sir Charles Bell determined that the seventh cranial nerve controlled the muscles of facial expression.
- A peripheral (lower motor neuron) facial palsy weakens all the ipsilateral facial muscles, including the frontalis and orbicularis oculi. A central (upper motor neuron) facial palsy weakens only the contralateral two-thirds of the face, sparing the frontalis.

**FIGURE 5-1**

Sir Charles Bell (1774 to 1842), a native of Edinburgh, Scotland, who practiced in London, was a surgeon but is best remembered as an anatomist. His name is attached to facial palsy because he was the first to demonstrate, both experimentally and by clinical observation, that the muscles of facial expression were innervated by the seventh cranial nerve.

*monkey, the very peculiar activity of his features on that side ceased altogether. The timid motions of his eye-lids and eye-brows were lost, and he could not wink on that side; and his lips were drawn to the other side, like a paralytic drunkard, whenever he showed his teeth in rage...the conclusion is inevitable, that the motions of the lips, nostrils and eye-lids, and forehead, in expression, have nothing to do with the fifth pair of nerves... A man had the trunk of the respiratory nerve of the face injured by a suppuration, which took place anterior to the ear, and through which the nerve passed in its course to the face. It was observed, that in smiling and laughing, his mouth was drawn*

*in a very remarkable manner to the opposite side. The attempt to whistle was attended with a ludicrous distortion of the lips: when he took snuff and sneezed, the side where the suppuration had affected the nerve remained placid, while the opposite side exhibited the usual distortion.*<sup>3</sup>

Bell was born in Edinburgh, Scotland, and trained as a surgeon, but he is best recognized for his contributions as an anatomist. He was also an accomplished artist who illustrated many of his own dissections. In 1804 Bell moved to London, where he helped found medical schools at University College London and Middlesex. Prior to his discovery of the innervation of the face, Bell demonstrated that the ventral spinal root is motor; the Bell-Magendie law attributed motor and sensory functions to the ventral and dorsal roots, respectively. In addition to Bell's palsy and the Bell-Magendie law, he is also eponymized by Bell's phenomenon (upward deviation of the eyeball during forced closure of the lids) and by the long thoracic nerve of Bell that innervates the serratus anterior.<sup>4-6</sup>

**ANATOMY OF CRANIAL NERVE VII**

When evaluating a patient with a peripheral facial palsy, the most clinically relevant anatomic facts to appreciate are the following:

- A peripheral (lower motor neuron) facial palsy weakens the entire ipsilateral face, including the frontalis and orbicularis oculi muscles, which are spared with central (upper motor neuron) lesions that cause weakness of only the lower two-thirds of the contralateral face (**Figure 5-2**). The features of a peripheral facial palsy were beautifully described by Romberg<sup>7</sup>: "The patient is unable



**FIGURE 5-2** In the top images, both patients demonstrate weakness of the lower face with pulling of the mouth to the normal side when smiling. The sparing of the left frontalis in the left lower image confirms that this is upper motor neuron facial weakness, whereas the right frontalis cannot be contracted in the patient on the lower right, who has Bell's palsy.

to corrugate his forehead, the furrows on which disappear at once with the paralysis, so that the brow of an old man becomes as smooth as that of a child, and no more effectual cosmetic is to be found for elderly ladies.” Therefore, a helpful clinical pearl for differentiating a central versus peripheral facial palsy is that the latter “gets out the wrinkles.” Of course, this is not clinically useful in the young, unwrinkled patient.

- It is distinctly rare to have a parenchymal lesion affect the facial nucleus or fascicle in isolation. The clue that a peripheral facial palsy is from a central lesion is the presence of neighborhood signs localizing to the pons. These include an ipsilateral horizontal gaze palsy due to involvement of the paramedian pontine reticular formation; ipsilateral sixth nerve palsy; internuclear ophthalmoplegia (INO) due to involvement of the



**KEY POINT**

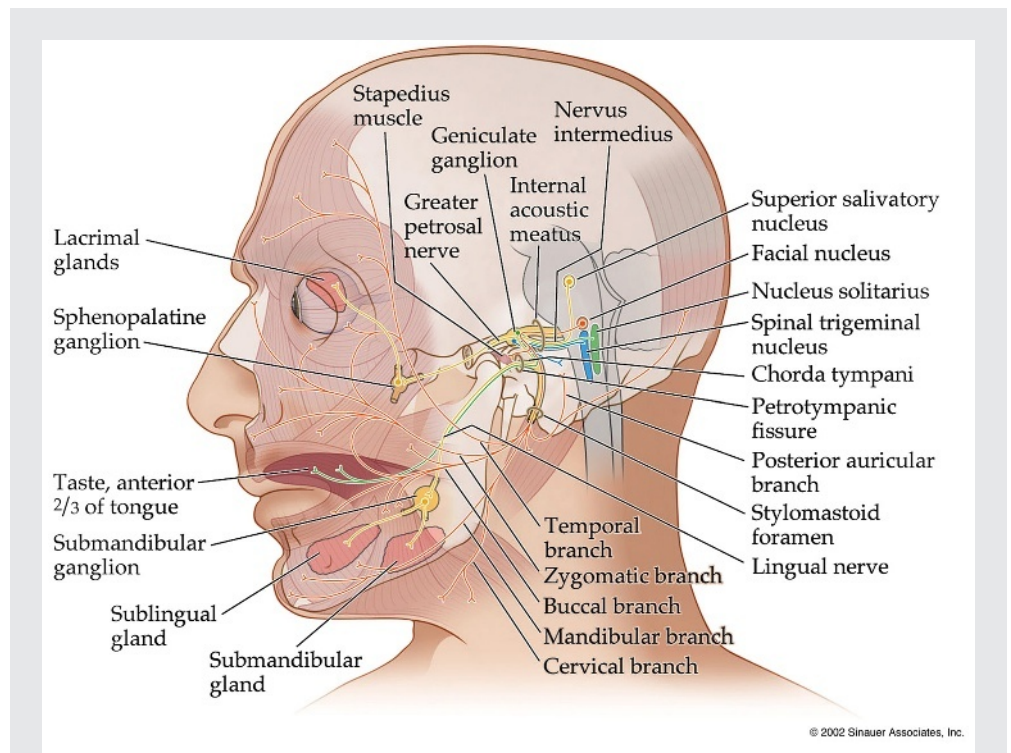
■ In addition to innervating the muscles of facial expression, the facial nerve also conveys sensation from the external auditory canal, pinna, mastoid, and mucosa of the palate; innervates the stapedius muscle and the lacrimal and minor salivary glands; and carries taste from the anterior two-thirds of the tongue.

ipsilateral medial longitudinal fasciculus; ipsilateral facial numbness due to involvement of the descending tract of cranial nerve V; and a contralateral hemiparesis. The combination of a peripheral seventh cranial nerve palsy along with an ipsilateral horizontal gaze palsy and INO is known as the eight-and-a-half syndrome, adding the seventh nerve palsy to the more well-known one-and-a-half syndrome.<sup>8</sup>

- The facial nerve innervates more than just the muscles of facial expression. Afferent fibers convey sensation from the external auditory canal, pinna, mastoid, and mucosa of the palate (hence the need to check the palate as well as the auditory canal for vesicles in suspected geniculate zoster/Ramsay

Hunt syndrome) as well as taste from the anterior two-thirds of the tongue. Parasympathetic fibers in cranial nerve VII innervate the lacrimal gland and minor salivary glands. The sensory and parasympathetic fibers are carried via the nervus intermedius (Wrisberg nerve) (Figure 5-3).<sup>1,10-12</sup>

The nucleus of cranial nerve VII is in the tegmentum of the caudal pons. The fascicle ascends, looping around the abducens nucleus, protruding in the fourth ventricle as the facial colliculus before exiting the dorsolateral pons (cerebellopontine angle). The parasympathetics arise in the superior salivatory nucleus; taste fibers terminate in the nucleus of the tractus solitarius, and the sensory afferents terminate in the nucleus of the spinal tract of cranial nerve V.



**FIGURE 5-3** The facial nerve and its branches.

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The facial nerve travels with the vestibulocochlear nerve in the internal auditory meatus before entering the facial canal (fallopian canal), a narrow bony canal within the temporal bone. It is because of its course through this narrow canal, with little room for expansion, that inflammation of the nerve (due to any cause) is thought to cause compression resulting in paralysis and, as will be discussed, is the rationale for the use of corticosteroids for Bell's palsy. The first branch of the facial nerve to exit, at the level of the geniculate ganglion, is composed of the fibers innervating the lacrimal gland, via the greater superficial petrosal nerve. If lacrimation is diminished in a peripheral facial palsy, it suggests a more proximal lesion. Distal to the geniculate ganglion, the fibers innervating the stapedius muscle exit (this explains why some patients with Bell's palsy may have hyperacusis).

The chorda tympani is the final branch of cranial nerve VII before it exits the skull at the stylomastoid foramen. The chorda tympani, as previously mentioned, conveys taste from the anterior two-thirds of the tongue (as such, taste may be affected in Bell's palsy and is sometimes the first symptom) and joins with the lingual nerve to innervate the minor salivary glands (their involvement is rarely evident in Bell's palsy). From the stylomastoid foramen, the facial nerve courses through the parotid gland before dividing into branches that innervate all of the muscles of facial expression as well as the buccinator.<sup>1,10-12</sup> It is at this level that individual branches of cranial nerve VII can be affected by infiltrative or compressive lesions, or trauma, and cause a partial lower motor neuron facial palsy with sparing of the frontalis. This is why it is important to do a careful head and neck examination in the patient with a peripheral facial palsy, with particular

attention to the parotid gland, cervical adenopathy, or skin lesions, the latter relevant to perineural invasion by squamous or other types of cancer.<sup>13</sup>

Sparing of the frontalis with a "central (upper motor neuron) facial" is traditionally thought to reflect bilateral supranuclear innervation from the primary motor cortex, with the lower two-thirds of the face receiving predominantly contralateral innervation. In contrast, studies in nonhuman primates suggest that either there is little corticobulbar innervation of the VII subnuclei innervating the frontalis<sup>14</sup> or that innervation is from cortical areas distinct from those supplying the lower two-thirds of the face.<sup>15,16</sup> A study in humans suggests that sparing of the orbicularis oculi by upper motor neuron lesions is due to its dual innervation from cortical regions supplied by both the middle cerebral artery and anterior cerebral artery in contrast to the lower face, where cortical innervation is supplied solely by the middle cerebral artery.<sup>17</sup>

## EPIDEMIOLOGY

The incidence rate of Bell's palsy is 20 to 40 out of 100,000 per year, and sexes are equally affected with an average age of onset of 40 years. The incidence rate is highest in those age 70 years and older. No difference exists in the side of the face affected, nor does there appear to be a seasonal predominance.<sup>18,19</sup> A number of "risk" factors have been reported for Bell's palsy, including diabetes mellitus and hypertension, with little definitive evidence; however, both have been associated with a worse prognosis for recovery as has older age, non-ear pain, complete palsy, and decreased tearing.<sup>18-20</sup> Although pregnancy is often cited as a risk factor for Bell's palsy, this was not found to be the case in the epidemiologic study from Rochester, Minnesota,

## KEY POINT

- Factors suggesting a worse prognosis for recovery of Bell's palsy include diabetes mellitus, hypertension, older age, complete paralysis, lack of improvement by 1 month, non-ear pain, and decreased tearing.

by Hauser and colleagues.<sup>18</sup> In this regard, Katz and colleagues<sup>21</sup> found that risk factors for Bell's palsy during pregnancy included chronic hypertension, maternal obesity, and severe preeclampsia, but Bell's palsy had no effect on perinatal outcome (Case 5-1).

#### ETIOLOGY OF BELL'S PALSY

The cause of Bell's palsy is not known and may not be the same in all in-

dividuals. Edema of the facial nerve within the narrow fallopian canal has been observed during decompressive surgery for Bell's palsy<sup>22</sup> consistent with MRI enhancement of the facial nerve in Bell's palsy.<sup>23</sup> The cause of the edema may be ischemia in predisposed patients, such as the elderly or those with diabetes mellitus or hypertension, akin to other known ischemic cranial neuropathies, including the

### Case 5-1

A 31-year-old woman presented with a history of Bell's palsy, which had been diagnosed 1 month before the visit when she was 32 weeks pregnant, and her pregnancy had otherwise been uncomplicated. Her first symptom had been that her taste was "funny." The following day she had noticed "drooping" of the right face and difficulty closing the right eye. By the next day, she had complete right facial paralysis. She was seen by her obstetrician and started on prednisone and valacyclovir. About 1 week after the prednisone was stopped, she experienced 3 to 4 days of pain around the right side of her head and jaw, which she described as severe and that resolved spontaneously. The weakness had improved when she presented for neurologic consultation. On examination, the patient had minimal asymmetry of her face at rest; while showing her teeth, no movement of the right side of the face occurred; she could barely approximate the lids when squeezing the eyes shut, and the frontalis muscle on the right was weak (Figure 5-4). She reported 80% return of function 6 months later.



**FIGURE 5-4** Patient with Bell's palsy from Case 5-1. *A*, At rest, the patient has slight widening of the right palpebral fissure. *B*, When attempting to smile, the patient experiences clear weakness of the right side of her face with pulling of her mouth to the left. *C*, When closing her eyes, the right lids cannot be completely approximated. *D*, When asked to look surprised, the right frontalis does not contract.

**Comment.** The teaching points of this case include: (1) Bell's palsy can occur during pregnancy, although it is not clear that this is an actual risk factor; (2) altered or diminished taste can be the initial symptom of Bell's palsy; (3) pain is not an uncommon feature of Bell's palsy, either at presentation or during recovery; and (4) regarding pregnancy, valacyclovir is category B, suggesting that it is probably safe to use during pregnancy.

abducens and oculomotor nerves.<sup>24</sup> But this would not account for the many young people with Bell's palsy, including children. Herpes simplex virus (HSV) type 1, according to Gilden,<sup>1</sup> "is probably the cause of most cases of Bell's palsy...[and] reflects virus reactivation from latency in the geniculate ganglion rather than primary infection." Supporting evidence includes the isolation of HSV DNA from endoneurial fluid in Bell's palsy<sup>25</sup>; increased salivary shedding of HSV DNA in patients versus controls<sup>26,27</sup>; polymerase chain reaction (PCR) evidence of HSV type 1 in the geniculate ganglia<sup>28,29</sup>; and an HSV type 1 experimental animal model of Bell's palsy.<sup>30,31</sup> Despite this evidence, Gilden<sup>1</sup> acknowledged that "how the virus damages the facial nerve is uncertain."<sup>32,33</sup>

## EVALUATION OF BELL'S PALSY

Patients with Bell's palsy typically develop facial weakness over 1 to 2 days. They may find that toothpaste, liquids, or food leak from the affected side of the mouth, that the eyelid does not close, or that it is more difficult to speak, which leads many patients to the emergency department for fear of a stroke.

The key first step in evaluating the patient is to determine whether the facial weakness is peripheral or central. Of note is that, while almost all patients with a hemiparesis from stroke have facial weakness, it is rarely the presenting symptom and is often noticed by others rather than the patient (Case 5-2). As discussed in the section on anatomy, with a *central facial* palsy, sparing of the upper one-third of the contralateral face occurs. With a *peripheral* facial palsy, weakness of *all* muscles of facial expression occurs. The furrow is lost from the brow, and the patient cannot elevate the brow (ie, in response to the

command, "raise your forehead like you're surprised"), the palpebral fissure is wider, the nasolabial fold is flattened, the cheek cannot be puffed out, and the nares do not flare with a hard inspiration. The patient is unable to whistle, and when smiling or showing teeth, the mouth is drawn to the intact side. Having the patient test his or her ability to whistle is a useful way to document recovery.

Although most patients with Bell's palsy do not notice a dry eye (recall that the lacrimal gland is innervated by cranial nerve VII), nevertheless, as discussed in the section on treatment, proper lubrication and eye care is necessary, particularly when severe weakness of the orbicularis oculi occurs to the extent that the upper and lower lids cannot be approximated. Paradoxically, some patients may present with tears running down the cheek, presumably due to weakness of the inferior portion of the orbicularis oculi, preventing tears from being directed toward the lacrimal duct, possibly in combination with ocular irritation. If the stapedius muscle is involved, hyperacusis may occur, as contraction of the stapedius functions to dampen the ossicles. Despite the facial nerve innervation of minor salivary glands, dry mouth is usually not experienced. Involvement of the chorda tympani causes loss of taste in the ipsilateral anterior two-thirds of the tongue; this can sometimes be the first symptom noticed by the patient, and impaired taste may portend a worse prognosis for recovery. Other factors reported to be associated with a worse outcome include complete facial palsy, older age, diabetes mellitus, and, as mentioned in the following section, non-ear pain.<sup>18–20</sup>

It is not uncommon for patients with Bell's palsy to report pain—typically around the ear, mastoid, and face—and

## KEY POINTS

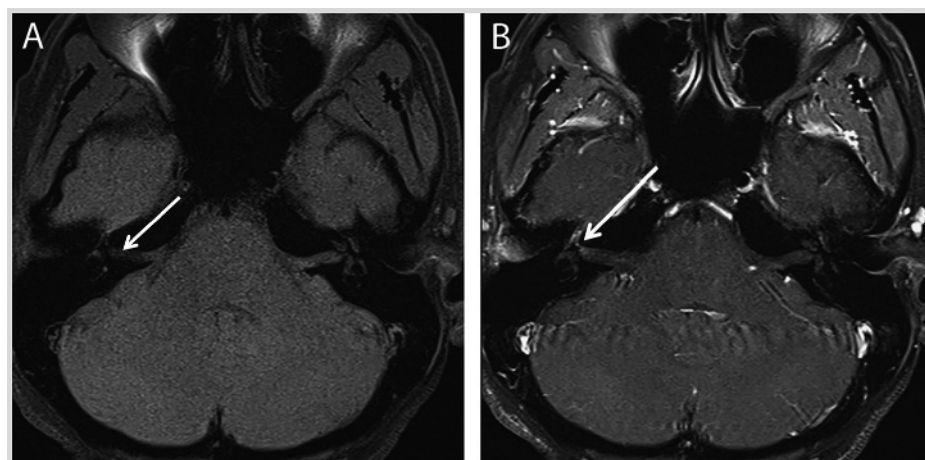
- The cause of Bell's palsy is not known but may be due to reactivation of herpes simplex virus type 1.
- The return of the ability to whistle can be a useful way to document improvement in Bell's palsy (assuming the patient could whistle before its onset).
- Although typically no objective sensory loss occurs with Bell's palsy, pain around the ear and face may occur, which at times can be severe. Prolonged pain outside of these areas may be a sign that the patient has an alternative cause of facial palsy.



**Case 5-2**

A 40-year-old woman presented to the emergency department after noticing that her face felt “swollen” upon awakening, and she noted that the right side of her face felt “distorted and weak.” She noticed toothpaste leaking from the right side of her mouth, tearing of the right eye, and inability to flair the right nostril.

On examination, the patient had mild right peripheral facial paresis with an otherwise normal examination. As she was initially suspected of having a stroke, she underwent an MRI, which demonstrated mild enhancement of the intracanalicular and labyrinthine segments of the right facial nerve (**Figure 5-5**). She was treated with eye care, corticosteroids, and valacyclovir. She had complete resolution of her symptoms by 3 months.

**FIGURE 5-5**

Axial MRI of patient in **Case 5-2**. *A*, Unenhanced MRI demonstrating a normal facial nerve (*arrow*). *B*, Enhancement of the intracanalicular and labyrinthine segments of the right facial nerve (*arrow*), which can be seen in Bell's palsy.

Courtesy of Robert Morales, MD.

**Comment.** Recognition that this patient had a peripheral facial palsy, in isolation, would have steered the diagnosis and evaluation from a stroke and toward Bell's palsy, obviating the need for the MRI. Nevertheless, it does confirm that enhancement of these particular segments of the facial nerve may occur in patients with Bell's palsy.

**KEY POINT**

■ Some have suggested that Bell's palsy is actually a cranial polyneuropathy. But, anything more than subtle signs implicating other cranial nerves should be viewed as a potential red flag, casting doubt on the diagnosis of Bell's palsy.

non-ear pain has been associated with a worse prognosis. Likewise, patients may report facial “numbness” (it is important to ask what they mean by numbness), but sensory testing is usually normal in Bell's palsy. Yet, some authors have suggested that patients with Bell's palsy in fact have a cranial polyneuropathy. Adour<sup>11</sup> found evidence of involvement of cranial nerves V, VIII, IX, and X in patients with Bell's palsy.<sup>34</sup> In a series of 51 patients diag-

nosed with Bell's palsy, Benatar and colleagues<sup>35</sup> found that 8% had evidence of involvement of at least one other cranial nerve, including the trigeminal, glossopharyngeal, and hypoglossal. These findings raise several questions: is Bell's palsy really a cranial polyneuropathy simply dominated by involvement of the facial nerve? Or, should signs of involvement of other cranial nerves “rule out” Bell's palsy? The important point is that, in an



otherwise “typical” case of Bell’s palsy, patients may have other subtle cranial nerve symptoms and signs, but their presence should be a red flag that the condition might not be Bell’s palsy, and careful follow-up is warranted.

The majority of patients with Bell’s palsy will not have a recurrence, but a recurrence happens in about 7%, either on the same or the opposite side.<sup>36–38</sup> A recurrence should prompt a careful search for an alternative cause, such as sarcoidosis<sup>39,40</sup> or other inflammatory or infiltrative disorders. The Melkersson-Rosenthal syndrome is another consideration for recurrent peripheral facial palsy; it is characterized by a fissured tongue and periodic lip or facial swelling,<sup>41</sup> but many patients do not have the entire triad; inspection of the tongue can be an important clue.<sup>42</sup>

By adhering to the definition of Bell’s palsy as a spontaneous, acute, unilateral, isolated peripheral facial palsy, and if no red flags suggest an alternative cause, then neither laboratory testing nor imaging is needed<sup>43</sup>; unfortunately, the majority of patients undergo imaging. MRI of patients with Bell’s palsy may show enhancement in the intracanalicular and labyrinthine segments of the facial nerve (Case 5-2),<sup>23,44</sup> and this should not necessarily suggest an alternative diagnosis. As pointed out by Sartoretti-Schefer and colleagues,<sup>44</sup> the normal facial nerve may show enhancement of the geniculate ganglion and the tympanic-mastoid segment. Electrodiagnostic studies have little role in the management of Bell’s palsy.<sup>43</sup> According to a clinical practice guideline from the American Academy of Otolaryngology—Head and Neck Surgery Foundation, based on level C evidence, electrodiagnostic studies are not recommended for patients with incomplete facial paralysis, most of whom will have a good recovery, but may be offered to the patient with

complete paralysis for prognostic purposes, although it is not likely to change management.<sup>43</sup>

## DIFFERENTIAL DIAGNOSIS AND RED FLAGS

A very long list of causes of peripheral facial palsy exists (Table 5-1). At least 50% of peripheral facial palsies are due to Bell’s palsy, and when no red flags from the history or examination (Table 5-2) suggest an alternative diagnosis, the percentage is no doubt even higher. Most of the causes listed in Table 5-1 should not be confused with Bell’s palsy as long as one adheres to the features of Bell’s palsy previously mentioned: spontaneous, onset over 72 hours, otherwise normal neurologic and systemic examination, improvement over several months, and no red flags.

Historic red flags casting doubt on the diagnosis of Bell’s palsy as the cause of a peripheral facial palsy include *gradual onset* over weeks to months, concomitant vertigo or hearing loss, constitutional symptoms, cancer, human immunodeficiency virus (HIV)<sup>45,46</sup> or risk factors for HIV, and features suggesting Lyme disease (eg, endemic area, known tick bite, skin rash).<sup>47–49</sup> Since the majority of patients with Bell’s palsy improve within several months, the lack of any improvement or a gradual progression from facial paresis to facial palsy should both raise a red flag. About 7% of patients with Bell’s palsy will have a recurrence,<sup>37–39</sup> but when this occurs, it should be considered a red flag, prompting an evaluation (to include imaging and a lumbar puncture) for another cause.

Some of the red flags from the neurologic examination include bilateral facial palsy,<sup>50</sup> involvement of other cranial nerves (as mentioned previously, the physician may see subtle signs of other cranial nerve involvement in Bell’s

### KEY POINTS

- About 7% of patients with Bell’s palsy will have a recurrence.
- For typical cases of Bell’s palsy, with no red flags from the history or examination, no further workup, including imaging, is necessary.
- If a patient with Bell’s palsy is imaged with MRI (typically not necessary), enhancement of the intracanalicular and labyrinthine segments of the facial nerve may be seen.
- Red flags casting doubt on the diagnosis of Bell’s palsy include gradual onset, involvement of other cranial nerves, concurrent vertigo or hearing loss, bilaterality, risk for Lyme disease or human immunodeficiency virus, and systemic cancer.

**TABLE 5-1** Differential Diagnosis of Peripheral Facial Palsy

► **Parenchymal Lesion (Pontine)**

- Multiple sclerosis
- Stroke
- Abscess
- Encephalitis<sup>a</sup>
- Neoplasm
- Other

► **Congenital**

- Möbius syndrome<sup>a</sup>
- Forceps trauma

► **Trauma**

- Basilar skull fracture
- Postoperative/iatrogenic

► **Extraaxial Neoplasm**

- Schwannoma
- Neuroma
- Meningioma
- Metastatic
- Cholesteatoma
- Parotid
- Perineural invasion
- Other

► **Meningitis**

- Bacterial (including tetanus)
- Viral<sup>a</sup>
  - Human immunodeficiency virus<sup>a</sup> (including initial seroconversion)
  - Zika virus<sup>a</sup>
  - Epstein-Barr virus
  - Polio virus
  - Other
- Fungal

**TABLE 5-1** *Continued*

Mycobacteria

Tuberculosis

Leprosy

Spirochete

Syphilis

Lyme disease<sup>a</sup>

Sarcoidosis<sup>a</sup>

Neoplastic<sup>a</sup>

► **Infectious (Nonmeningitic), Inflammatory, or Infiltrative**

Geniculate zoster (Ramsay Hunt syndrome)

Guillain-Barré syndrome<sup>a</sup>

Chronic inflammatory demyelinating polyradiculoneuropathy

Osteomyelitis of skull base

Otitis media

Parotitis

Mastoiditis

Amyloidosis<sup>a</sup>

Granulomatosis with polyangiitis

Polyarteritis nodosa

Sjögren syndrome

► **Other**

Idiopathic intracranial hypertension

Melkersson-Rosenthal syndrome

Paget disease

Hereditary neuropathy with liability to pressure palsy

Wernicke encephalopathy

Ethylene glycol ingestion

*Continued on page 457*

**TABLE 5-1 Differential Diagnosis of Peripheral Facial Palsy** *Continued from page 456*

▶ **Myopathy or Neuromuscular Junction disorders<sup>a</sup>**

Facioscapulohumeral muscular dystrophy

Oculopharyngeal dystrophy

Myotonic dystrophy

Myasthenia gravis

<sup>a</sup> Facial palsy may be bilateral.

palsy), hemiparesis, hearing loss or nystagmus, ataxia, horizontal gaze palsy, or systemic weakness. Important findings on the head and neck examination to look for include vesicles in the external canal, tympanic membrane or palate, cervical adenopathy, otitis media, parotid mass, skin cancer, fissured tongue, and facial swelling.<sup>41,42</sup> It is important to recognize that a peripheral facial palsy may be the first sign of an evolving disorder such as neurosarcoidosis, meningeal carcinomatosis, or Guillain-Barré syndrome.<sup>51</sup>

Several specific causes of peripheral facial palsy deserve highlighting. It is the most common neurologic manifestation of Lyme disease,<sup>47-49</sup> and this should always be considered in patients who live in an endemic area, especially if the facial palsy is bilateral or in a child. Halperin and Golightly<sup>49</sup> determined that 25% of facial palsies during the summer months in an endemic area were due to Lyme disease, based on serology. Because no other clinical features may suggest Lyme disease before or at the time of facial palsy (eg, erythema migrans), and serologic evidence may lag behind the earliest clinical manifestations,<sup>47</sup> the decision to treat rests largely on the phy-

sician's index of suspicion for Lyme disease. If no symptoms or signs occur other than facial palsy, such as cranial polyneuropathy or evidence of parenchymal involvement, then a lumbar puncture is probably not indicated.<sup>48</sup> Even without antibiotics, the prognosis of Lyme disease facial palsy is favorable,<sup>52</sup> and therefore treatment is aimed at preventing sequelae. As pointed out by the American Academy of Neurology (AAN) Practice parameter, "no definitive data exist to establish the superiority, or lack thereof, of either oral or parenteral treatment."<sup>53</sup> For facial palsy presumed to be from

**TABLE 5-2 Red Flags Casting Doubt on the Diagnosis of Bell's Palsy**

- ▶ Gradual onset
- ▶ Vertigo, hearing loss, tinnitus
- ▶ No improvement within 3 months
- ▶ Bilateral facial palsy
- ▶ Other cranial nerve involvement
- ▶ Limb or bulbar weakness
- ▶ Parotid gland enlargement
- ▶ Otitis media
- ▶ Vesicles in external auditory canal, tympanic membrane, or oropharynx
- ▶ Cervical adenopathy
- ▶ Facial swelling/fissured (scrotal) tongue
- ▶ Skin rash/other signs of Lyme disease or living in an endemic area
- ▶ Risk factors for human immunodeficiency virus
- ▶ Facial skin cancer
- ▶ Systemic cancer

**KEY POINT**

■ Lyme disease is a common cause of peripheral facial palsy in endemic areas during the summer months, but in the absence of potential exposure and without other characteristic signs, routine testing is not recommended.

**KEY POINT**

■ A careful head and neck examination is important in patients with a peripheral facial palsy with particular attention for vesicles on the tympanic membrane, external auditory canal, or soft palate, indicating herpes zoster (Ramsay Hunt syndrome).

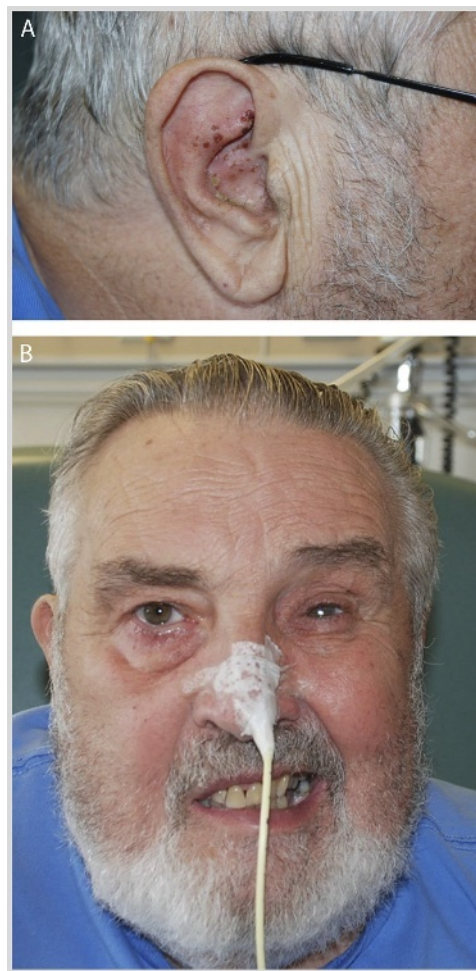
Lyme disease, it is reasonable to use an oral agent. If no clinical or serologic evidence confirms the diagnosis of Lyme disease at presentation, then steroids may be considered in case the diagnosis is actually Bell's palsy; according to the AAN practice parameter, although evidence is limited, a short course of steroids is not likely to be harmful when treating Lyme disease.<sup>53</sup>

Ramsay Hunt syndrome (Case 5-3) is manifested by a peripheral facial palsy with erythema and vesicles in the external auditory canal, the tympanic membrane, or the oropharynx. It is due to reactivation of varicella-zoster virus in the geniculate ganglion.<sup>54</sup> Accompanying symptoms reflect neighborhood involvement of the vestibulocochlear nerve including vertigo, hearing loss, and tinnitus.

**Case 5-3**

A 76-year-old man noticed ear pain followed by weakness of the right face. He did not experience hearing loss or vertigo. On examination, he had a right peripheral facial palsy and erythema and vesicles in the right ear (Figure 5-6). He was diagnosed as having geniculate zoster, Ramsay Hunt syndrome, and was treated with corticosteroids and acyclovir.

**Comment.** Although the involvement of the ear by zoster was readily apparent in this patient, in others it is subtle, necessitating careful inspection of the external auditory canal and tympanic membrane as well as the oropharynx. The presence of vertigo, hearing loss, and tinnitus may accompany Ramsay Hunt syndrome and are important red flags pointing away from Bell's palsy.

**FIGURE 5-6**

Images of patient in Case 5-3. *A*, The patient has erythema in the right auricle along with vesicles. *B*, When attempting to smile, the patient shows weakness of the right side of his face as well as less wrinkling of the right frontalis.



About 5% of patients with sarcoidosis will have neurologic involvement, and in one-half of those cases, it is the presenting sign, with a peripheral facial palsy being the most common manifestation. Neurosarcoidosis is particularly important to consider with bilateral peripheral facial palsy. The diagnosis may be challenging and requires a careful search for evidence of systemic involvement and, ideally, pathologic confirmation.<sup>39,40</sup> Another important consideration, particularly when bilateral facial palsy occurs, is Guillain-Barré syndrome, which may evolve rapidly from facial palsy into a more classic picture with bulbar and limb weakness with areflexia, but a subtype of Guillain-Barré syndrome manifests by only bifacial weakness and distal paresthesia.<sup>51</sup>

## MANAGEMENT

Most patients with Bell's palsy are worried they have had a stroke, so reassurance and education are important parts of treatment along with counseling that the prognosis is favorable for most patients, particularly those with mild or moderate facial weakness at presentation. Because of weakness of eyelid closure and the risk of corneal exposure, particularly in those with complete palsy, patients should use lubricating drops frequently during waking hours and a lubricating ointment at night. The lid can be taped closed at night or a cellophane patch can be applied with care so that no direct contact with the cornea occurs.

The use of steroids and antivirals to improve the recovery of Bell's palsy is based on the previously mentioned observations about the possible roles of edema and HSV type 1 in the pathophysiology and etiology, respectively, in Bell's palsy. Here the author relies primarily on the AAN evidence-based guidelines. The first guideline pub-

lished in 2001 found only five published studies of sufficient rigor upon which to determine the effectiveness of corticosteroids (two Class I, two Class II, and one Class III), and none were sufficiently powered to allow for a definitive recommendation.<sup>55</sup> By pooling the results of the Class I and II studies, coupled with the safety of a short course of steroids, the authors of this guideline concluded that: "steroids are safe and *probably* effective in improving facial functional outcomes in patients with Bell's palsy" (level B recommendation).<sup>55</sup> Even less evidence, and no Class I studies, existed upon which to base a recommendation for acyclovir, which received a level C recommendation of being safe and *possibly* effective. The lack of unbiased studies and a high complication rate precluded any evidence-based recommendations on surgical decompression.<sup>55</sup>

The AAN evidence-based guideline on steroids and antivirals for the treatment of Bell's palsy was updated in 2012,<sup>56</sup> and the revised recommendations were based largely on two Class I studies published since the original review. The first study by Sullivan and colleagues<sup>57</sup> compared four treatment arms: prednisolone alone (25 mg twice per day for 10 days), acyclovir alone (400 mg twice per day for 10 days), a combination of prednisolone and acyclovir, and placebo in patients age 16 or older who presented within 72 hours of onset of Bell's palsy (**Figure 5-7**). The primary outcome was the degree of facial weakness observed in digital photographs by three blinded reviewers, using the House-Brackmann facial nerve grading system, at 3 and 9 months. Of 551 patients who underwent randomization, outcome data were available for 496 (90%).

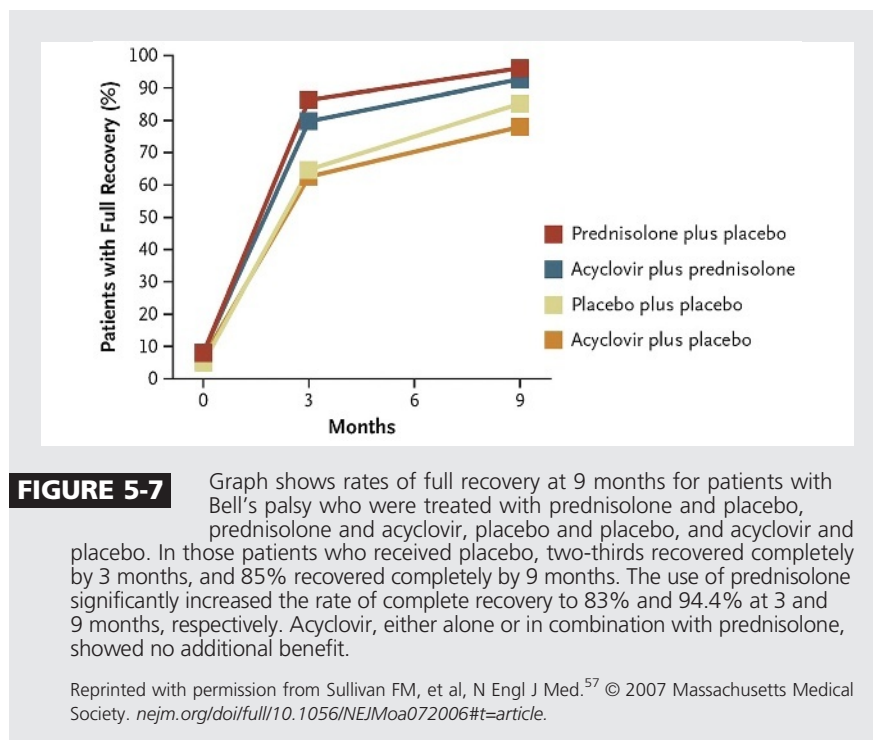
The other Class I study by Engström and colleagues<sup>58</sup> was similar, but instead of acyclovir, valacyclovir (1000 mg

## KEY POINTS

- A peripheral facial palsy is the most common neurologic manifestation of neurosarcoidosis and is particularly important to consider if bilateral.
- A peripheral facial palsy may be the first sign of Guillain-Barré syndrome, progressing to limb weakness, but a variant of Guillain-Barré syndrome occurs with only bifacial palsy.
- To protect the cornea in patients with Bell's palsy, lubricating drops should be used frequently during the day, and a lubricating gel should be used at night along with taping the lid closed or using a patch that does not rest on the cornea.

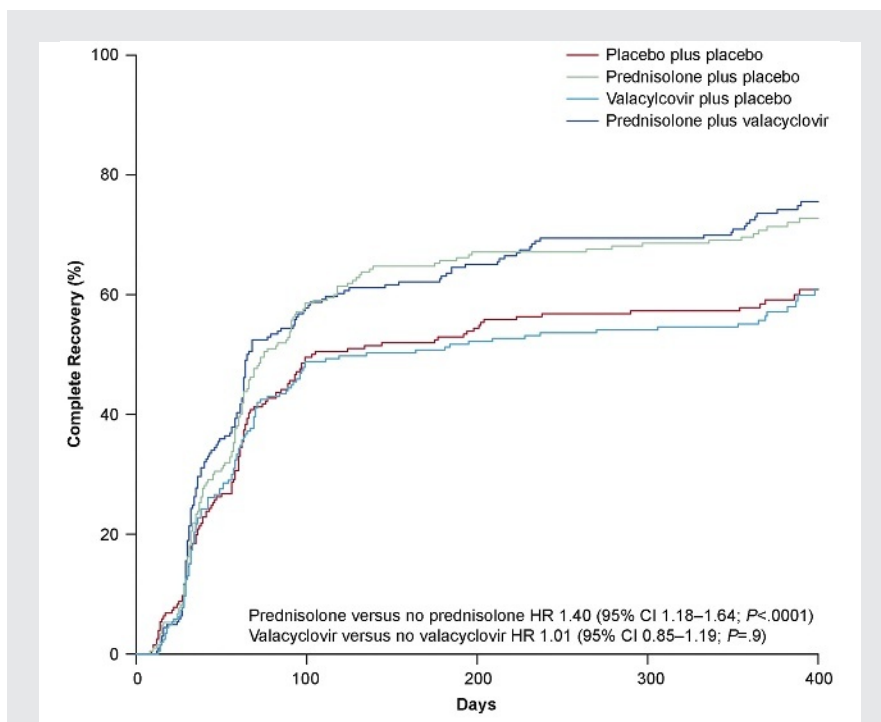
**KEY POINT**

■ The American Academy of Neurology evidence-based review gave a level A recommendation to the use of corticosteroids within the first 72 hours of onset to increase the likelihood of improvement for Bell's palsy. Combining an antiviral with a corticosteroid does not significantly improve the outcome of Bell's palsy, and their use was given a level C recommendation for a possible modest benefit, at best.



3 times per day for 7 days) was used. More than 800 patients between ages 18 and 75, presenting within 72 hours, were randomly assigned to prednisolone (60 mg/d for 5 days then reduced by 10 mg/d) and placebo, valacyclovir and placebo, placebo and placebo, and prednisolone and valacyclovir. The primary outcome measure was time to complete recovery (score of 100) on the Sunnybrook facial nerve grading system. Similar to the study by Sullivan and colleagues,<sup>57</sup> those patients who received prednisolone had a significantly higher rate of recovery at all time points, including the final assessment at 1 year compared to placebo and to the combination of valacyclovir and prednisolone (Figure 5-8). The lower rates of recovery, even for the placebo-placebo group in the study by Engström and colleagues,<sup>58</sup> were attributed to the higher sensitivity of the Sunnybrook scale for residual weakness. No significant adverse effects occurred in either study.

An editorial following the publication of these two trials by Tyler<sup>32</sup> concluded that “antiviral therapy should not be routinely used in patients with [Bell's palsy].” Tyler did, however, point out the potential benefit of antivirals for treatment of Ramsay Hunt syndrome.<sup>32</sup> The AAN evidence-based review gave a level A recommendation to the use of corticosteroids for treatment of Bell's palsy within 72 hours of onset. Continuing, they reiterated that adding an antiviral to a corticosteroid offers no significant benefit regarding facial recovery. However, they pointed out that the 95% confidence interval of the two Class I studies could not rule out a modest effect of adding an antiviral and gave a Class C rating to the combination.<sup>56</sup> When discussing the combination with patients, despite counseling that adding an antiviral to a steroid does not offer significant benefit (when also told that a benefit, even modest at best, cannot be “ruled out”), most



**FIGURE 5-8** Percent of patients with Bell's palsy making a complete recovery (Sunnybrook scale score of 100) by time and treatment groups. Patients who received prednisolone and placebo had a significantly faster rate of recovery of complete facial function (75 days) compared to those receiving placebo-placebo (104 days) or valacyclovir-placebo (135 days). Similarly, a significantly higher rate of recovery was seen at 3, 6, and 12 months in patients who received prednisolone. No significant additional improvement in the recovery rate was shown when prednisolone was combined with valacyclovir.

CI = confidence interval; HR = hazard ratio.

Reprinted with permission from Engström M, et al, *Lancet Neurol*.<sup>58</sup> © 2008 Elsevier. [thelancet.com/journals/laner/article/PIIS1474-4422\(08\)70221-7/abstract](http://thelancet.com/journals/laner/article/PIIS1474-4422(08)70221-7/abstract).

patients, in the author's experience, opt to take an antiviral.

The clinical practice guidelines by the American Academy of Otolaryngology—Head and Neck Surgery Foundation, developed by specialists in a variety of related fields including neurology, made similar recommendations about the use of corticosteroids and antivirals for Bell's palsy. They went on to address some of the other popularly touted treatments for Bell's palsy, finding insufficient or poor-quality evidence to make any recommendations about the use of surgical decompression, acupuncture, and physical therapy to treat Bell's palsy.<sup>43</sup>

## LONG-TERM MANIFESTATIONS AND COMPLICATIONS

As the placebo arms of the previously mentioned studies demonstrate, up to 85% of patients with Bell's palsy make a complete recovery within 1 year.<sup>57,58</sup> Those left with facial weakness may be candidates for surgical procedures aimed at improving facial function, particularly when significant eyelid weakness occurs, with risk of exposure keratitis (this complication requires prompt ophthalmologic referral). These include implantation of a gold weight in the eyelid and other nerve or muscle transfer procedures that will not be reviewed here; patients should be

**KEY POINT**

■ Residual facial weakness after Bell's palsy can cause a contracture, making it appear at rest that the normal side is weak with a flatter nasolabial fold and widened palpebral fissure. When facial function is tested, the weak side is readily apparent.

referred to a surgeon experienced in these options.<sup>13,59</sup>

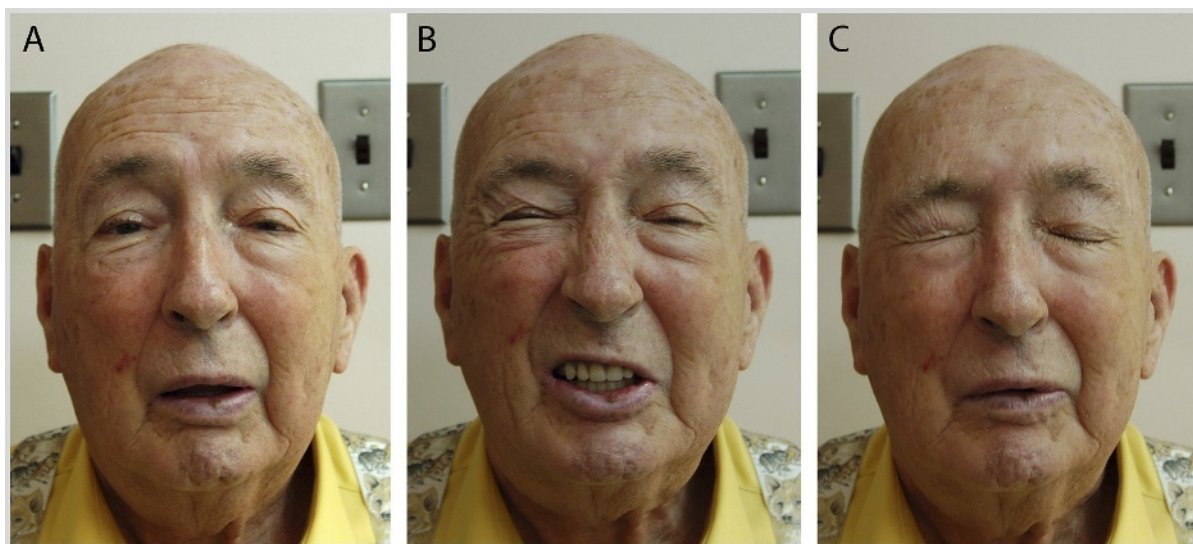
When residual weakness occurs in patients with Bell's palsy, the apparent side of the weakness can be paradoxical. With an acute peripheral facial palsy, the nasolabial fold is flattened, and the palpebral fissure is widened. But with chronic weakness, a contracture may develop such that at rest, the nasolabial fold on the weak side is deeper and the palpebral fissure narrower (**Case 5-4**).<sup>60</sup> The actual side of the weakness is readily apparent with movement, and synkinesia almost al-

ways occurs, confirming an old facial palsy. The author has been involved in a few cases where an old case of Bell's palsy with contracture has been misinterpreted as new weakness on the patient's good side of the face, leading to an erroneous diagnosis (usually stroke) and unnecessary testing before the findings are interpreted correctly and the history clarified. Patients are often unaware of residual weakness and synkinesia and may forget about having had Bell's palsy many years earlier.

There are two types of synkinesias that develop after Bell's palsy due to

**Case 5-4**

A 73-year-old man was seen for Parkinson disease and related a history of remote Bell's palsy, from which he reported a good recovery. On examination, at rest (**Figure 5-9A**) the right nasolabial fold was flatter, and the palpebral fissure was wider, suggesting that was the side of the facial weakness. However, as **Figures 5-9B** and **5-9C** demonstrate, he had a contracture on the left, which is the side of the weakness.

**FIGURE 5-9**

Images of the patient in **Case 5-4**. When comparing the sides of the patient's face at rest, there is flattening of the right nasolabial fold and widening of the right palpebral fissure (**A**), suggesting right-sided facial weakness. But, when smiling (**B**) or closing his eyes (**C**), it is apparent that the weakness is present on the left. The asymmetry at rest reflects contracture of the left face from a remote Bell's palsy.

Panel A reprinted with permission from Reich SG, *Neurology*.<sup>60</sup> © 2007 American Academy of Neurology. [neurology.org/content/68/2/E1.full](http://neurology.org/content/68/2/E1.full).

**Comment.** This case demonstrates that it can be easy to mistake the side of weakness from a previous case of Bell's palsy, but the synkinesia is a sure giveaway.



misdirection of regenerating fibers. The first is motor: with blinking, particularly if forceful, simultaneous contracture of the ipsilateral mouth or platysma occurs, and similarly, when smiling or showing teeth, contracture of the orbicular oculi and narrowing of the palpebral fissure occurs (Case 5-5). This is often asymptomatic but if patients are bothered, then botulinum toxin is the most effective treatment. Some patients go on to develop hemifacial spasm.<sup>61</sup>

Nonmotor fibers in the facial nerve may also misdirect, producing two types of synkinesias. The first is gustatory tearing in which salivation may be associated with lacrimation (so-called crocodile tears named for the myth that the crocodile either sheds a tear to attract its victim or sheds a tear as the victim is being eaten—in either case, the implication is that crocodile tears are insincere tears).

When salivation causes facial sweating, this is known as Frey syndrome or gustatory sweating, named for Lucja Frey, one of the first female Polish neurologists, whose productive career was cut tragically short by the Nazis.<sup>62</sup> Although Frey was not the first to recognize this phenomenon, she is credited with discerning its physiology and pharmacology. Frey syndrome is

encountered much more commonly after parotid surgery than Bell's palsy (see the video from Reich and Grill<sup>62</sup> for an example of Frey syndrome).

## CONCLUSION

Bell's palsy is characterized by the spontaneous, acute (over 24 to 72 hours) onset of unilateral peripheral facial palsy in isolation—that is, no features from the history, neurologic examination, or general examination suggest an alternative diagnosis. Red flags casting doubt on the diagnosis of Bell's palsy include gradual onset, concurrent vertigo or hearing loss, vesicles in the external auditory canal, living in an area endemic for Lyme disease, risk factors for HIV, and systemic cancer, among others. When no red flags exist, and the diagnostic criteria for Bell's palsy are used, additional testing is usually not necessary. Even without treatment, most patients, especially those with facial paresis rather than palsy, will have a complete recovery. A 10-day course of corticosteroids has been shown to significantly increase the likelihood of recovery; adding an antiviral does not significantly improve the likelihood of recovery, but the possibility that doing so may have a modest benefit, at most, cannot be ruled out. Residual effects of

## KEY POINTS

- A motor synkinesia is a complication of Bell's palsy. This is usually asymptomatic but, if bothersome, can be treated with botulinum toxin.
- Two nonmotor synkinesias after Bell's palsy include gustatory lacrimation (crocodile tears) and gustatory sweating, known as Frey syndrome.

## Case 5-5

A 55-year-old woman had undergone surgery for a large right acoustic neuroma 20 years earlier and was followed by her neurologist for headaches. Postoperatively, she had had moderate facial weakness that gradually improved, and she eventually developed an asymptomatic synkinesia. When she blinked, simultaneous movement of the right lips and mentalis occurred, and when she smiled, contraction of the orbicularis oculi occurred (Supplemental Digital Content 5-1, [links.lww.com/CONT/A214](http://links.lww.com/CONT/A214)).

**Comment.** This case demonstrates one of the complications of facial nerve palsy: the aberrant regeneration of motor fibers. The patient exhibits synkinesia between the orbicularis oculi and orbicularis oris, so that moving the mouth causes a contraction, which narrows the palpebral fissure, and eye closure causes retraction of the mouth. This symptom is often asymptomatic but, if bothersome, it can be treated with botulinum toxin.

Bell's palsy include permanent weakness and motor and nonmotor synkinesia, the latter including gustatory tearing and gustatory sweating (Frey syndrome).

### VIDEO LEGEND Supplemental Digital Content 5-1

**Synkinesia after right facial nerve palsy.** The 55-year-old woman in **Case 5-5** developed a moderate right facial nerve palsy 20 years earlier as a consequence of the removal of a large acoustic neuroma, which gradually improved. When she blinks, co-contraction of the right orbicularis oris occurs, and when she smiles, co-contraction of the right orbicularis oculi occurs, causing the palpebral fissure to narrow; these are signs of aberrant regeneration of the facial nerve with synkinesia.

*links.lww.com/CONT/A214*

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### ACKNOWLEDGMENT

This article is dedicated to Donald H. Gilden, MD, FAAN (1937 to 2016), in recognition of his many contributions to Bell's palsy and with appreciation of his friendship.

### REFERENCES

- Gilden DH. Clinical practice. Bell's palsy. *N Engl J Med* 2004;351(13):1323–1331. doi:10.1056/NEJMcp041120.
- Sajadi MM, Sajadi MR, Tabatabaie SM. The history of facial palsy and spasm: Hippocrates to Razi. *Neurology* 2011;77(2):174–178. doi:10.1212/WNL.0b013e3182242d23.
- Bell C. On the nerves: giving an account of some experiments on their structure and functions, which lead to a new arrangement of the system. *Phil Trans R Soc Lond* 1821;111:398–424.
- Rose FC. A short history of neurology: the British contribution 1660-1910. Woburn, MA: Butterworth-Heinemann 1999;42–46:122–128.
- Sir Charles Bell. NNDB. *nndb.com/people/118/000100815*. Accessed January 30, 2017.
- Van Gijn J. Charles Bell (1774-1842). *J Neurol* 2011;258(6):1189–1190. doi:10.1007/s00415-011-5912-5.
- Romberg MH. Chapter XXXV: paralysis of the facial nerve. In: Sieveking EH, ed. *A manual of the nervous diseases of man*. London, UK: Sydenham Society, 1853:267–286.
- Eggenberger E. Eight-and-a-half syndrome: one-and-a-half syndrome plus cranial nerve VII palsy. *J Neuroophthalmol* 1998;18(2):114–116.
- Blumenfeld H. *Neuroanatomy through clinical cases*. 2nd ed. Sunderland, MA: Sinauer Associates, Inc, 2010.
- Brazis PW, Masdeau JC, Biller J. Cranial nerve VII (the facial nerve). In: *Localization in clinical neurology*. Philadelphia: Lippincott Williams & Wilkins, 2001:289–397.
- Adour KK. Current concepts in neurology: diagnosis and management of facial paralysis. *N Engl J Med* 1982;307(6):348–351.
- Gilchrist JM. Seventh cranial neuropathy. *Semin Neurol* 2009;29(1):5–13. doi:10.1055/s-0028-1124018.
- Warren TA, Nagle CM, Bowman J, Panizza BJ. The natural history and treatment outcomes of perineural spread of malignancy within the head and neck. *J Neurol Surg B Skull Base* 2016;77(2):107–112. doi:10.1055/s-0036-1579777.
- Jenny AB, Saper CB. Organization of the facial nucleus and corticofacial projection in the monkey: a reconsideration of the upper motor neuron facial palsy. *Neurology* 1987;37(6):930–939. doi:10.1212/WNL.37.6.930.
- Morecraft RJ, Stilwell-Morecraft KS, Rossing WR. The motor cortex and facial expression: new insights from neuroscience. *Neurologist* 2004;10(5):235–249.
- Morecraft RJ, Louie JL, Herrick JL, Stilwell-Morecraft KS. Cortical innervation of the facial nucleus in the non-human primate: a new interpretation of the effects of stroke and related subtotal brain trauma on the muscles of facial expression. *Brain* 2001;124(pt 1):176–208. doi:10.1093/brain/124.1.176.
- Cattaneo L, Sacconi E, De Giampaulis P, et al. Central facial palsy revisited: a clinical-radiological study. *Ann Neurol* 2010;68(3):404–408. doi:10.1002/ana.22069.
- Hauser WA, Karnes WE, Annis J, Kurland LT. Incidence and prognosis of Bell's palsy in the population of Rochester, Minnesota. *Mayo Clin Proc* 1971;46(4):258–264.

19. Katusic SK, Beard CM, Wiederholt WC, et al. Incidence, clinical features, and prognosis in Bell's palsy, Rochester, Minnesota 1968-1982. *Ann Neurol* 1986;20(5):622-627.
20. Adour KK, Wingerd J. Idiopathic facial paralysis (Bell's palsy): factors affecting severity and outcome in 446 patients. *Neurology* 1974;24(12):1112-1116.
21. Katz A, Sergienko R, Dior U, et al. Bell's palsy during pregnancy: is it associated with adverse perinatal outcome? *Laryngoscope* 2011;121(7):1395-1398. doi:10.1002/lary.21860.
22. Cawthorne T. The pathology and surgical treatment of Bell's palsy. *Proc R Soc Med* 1951;44(7):565-572.
23. Yetiser S, Kazkayas M, Altinok D, Karadeniz Y. Magnetic resonance imaging of the intratemporal facial nerve in idiopathic peripheral facial palsy. *Clin Imaging* 2003;27(2):77-81. doi:10.1016/S0899-7071(02)00485-0.
24. Kung NH, Van Stavern GP. Isolated ocular motor nerve palsies. *Semin Neurol* 2015;35(05):539-548. doi:10.1055/s-0035-1563568.
25. Murakami S, Mizobuchi M, Nakashiro Y, et al. Bell palsy and herpes simplex virus: identification of viral DNA in endoneurial fluid and muscle. *Ann Intern Med* 1996;124(1 pt 1):27-30. doi:10.7326/0003-4819-124-1\_Part\_1-199601010-00005.
26. Furuta Y, Fukuda S, Chida E, et al. Reactivation of herpes simplex virus type 1 in patients with Bell's palsy. *J Med Virol* 1998;54(3):162-166.
27. Abiko Y, Ikeda M, Hondo R. Secretion and dynamics of herpes simplex virus in tears and saliva of patients with Bell's palsy. *Otol Neurotol* 2002;23(5):779-783.
28. Burgess RC, Michaels L, Bale JF Jr, Smith RJH. Polymerase chain reaction amplification of herpes simplex viral DNA from the geniculate ganglion of a patient with Bell's palsy. *Ann Otol Rhinol Laryngol* 1994;103(10):775-779. doi:10.1177/000348949410301006.
29. Takasu T, Furuta Y, Sato KC, et al. Detection of latent herpes simplex virus DNA and RNA in human geniculate ganglia by the polymerase chain reaction. *Acta Otolaryngol* 1992;112(6):1004-1011.
30. Takahashi H, Hitsumoto Y, Honda N, et al. Mouse model of Bell's palsy induced by reactivation of herpes simplex virus type 1. *J Neuropathol Exp Neurol* 2001;60(6):621-627. doi:10.1093/jnen/60.6.621.
31. Sugita T, Murakami S, Yanagihara N, et al. Facial nerve paralysis induced by herpes simplex virus in mice: an animal model of acute and transient facial paralysis. *Ann Otol Rhinol Laryngol* 1995;104(7):574-581.
32. Tyler KL. Prednisone—but not antiviral drugs—improves outcome in patients with Bell's palsy. *Nat Rev Neurol* 2009;5:74-75. doi:10.1038/ncpneuro1002.
33. Kennedy PG. Herpes simplex virus type 1 and Bell's palsy—a current assessment of the controversy. *J Neurovirol* 2010;16(1):1-5. doi:10.3109/13550280903552446.
34. Adour KK, Byl FM, Hilsinger RL Jr, et al. The true nature of Bell's palsy: analysis of 1,000 consecutive patients. *Laryngoscope* 1978;88(5):787-801.
35. Benatar M, Edlow J. The spectrum of cranial neuropathy in patients with Bell's palsy. *Arch Intern Med* 2004;164(21):2383-2385. doi:10.1001/archinte.164.21.2383.
36. Hohman MH, Hadlock TA. Etiology diagnosis and management of facial palsy: 2000 patients at a facial nerve center. *Laryngoscope* 2014;125(7):E283-E293. doi:10.1002/lary.24542.
37. Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Otolaryngol Suppl* 2002;(549):4-30. doi:10.1080/000164802760370736.
38. Pitts DB, Adour KK, Hilsinger RL Jr. Recurrent Bell's palsy: analysis of 140 patients. *Laryngoscope* 1988;98(5):535-540.
39. Stern BJ, Krumholz A, Johns C, et al. Sarcoidosis and its neurological manifestations. *Arch Neurol* 1985;42(9):909-917.
40. Bagnato F, Stern BJ. Neurosarcoidosis: diagnosis, therapy and biomarkers. *Expert Rev Neurother* 2015;15(5):533-548. doi:10.1586/14737175.2015.1037288.
41. Levenson MJ, Ingerman M, Grimes C, Anand KV. Melkersson-Rosenthal syndrome. *Arch Otolaryngol* 1984;110(8):540-542.
42. Greene RM, Rogers RS 3rd. Melkersson-Rosenthal syndrome: a review of 36 patients. *J Am Acad Dermatol* 1989;21(6):1263-1270.
43. Baugh RF, Basura GJ, Ishii LE, et al. Clinical practice guideline: Bell's palsy executive summary. *Otolaryngol Head Neck Surg* 2013;149(5):656-663. doi:10.1177/0194599813506835.
44. Sartoretti-Schefer S, Wichmann W, Valavanis A. Idiopathic, herpetic, and HIV-associated facial nerve palsies: abnormal MR enhancement patterns. *AJNR Am J Neuroradiol* 1994;15(3):479-485.

45. Riancho J, Delgado-Alvarado M, Valero C, et al. Clinical spectrum of peripheral facial paralysis in HIV-infected patients according to HIV status. *Int J STD AIDS* 2013;24(1):39–41. doi:10.1177/0956462412472308.
46. Serrano P, Hernández N, Arroyo JA, et al. Bilateral Bell palsy and acute HIV type 1 infection: report of 2 cases and review. *Clin Infect Dis* 2007;44(6):e57–e61. doi:10.1086/511876.
47. Halperin JJ. Nervous system Lyme disease. *Curr Infect Dis Rep* 2015;17(1):445. doi:10.1007/s11908-014-0445-6.
48. Halperin JJ. Facial nerve palsy associated with Lyme disease. *Muscle Nerve* 2003;28(4):516–517. doi:10.1002/mus.10451.
49. Halperin JJ, Golightly M. Lyme borreliosis in Bell's palsy. Long Island Neuroborreliosis Collaborative Study Group. *Neurology* 1992;42(7):1268–1270.
50. Keane JR. Bilateral seventh nerve palsy: analysis of 43 cases and review of the literature. *Neurology* 1994;44(7):1198–1202.
51. Wakerley BR, Yuki N. Isolated facial diplegia in Guillian-Barré syndrome: bifacial weakness with paresthesias. *Muscle Nerve* 2015;52(6):927–932. doi:10.1002/mus.24887.
52. Clark JR, Carlson RD, Sasaki CT, et al. Facial paralysis in Lyme disease. *Laryngoscope* 1985;95(11):1341–1345.
53. Halperin JJ, Shapiro ED, Logigian E, et al. Practice parameter: treatment of nervous system Lyme disease (an evidence-based review). Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2007;69(1):91–102. doi:10.1212.01.wnl.0000265517.66976.28.
54. Sweeney CJ, Gilden DH. Ramsay Hunt syndrome. *J Neurol Neurosurg Psychiatry* 2001;71(2):149–154. doi:10.1136/jnnp.71.2.149.
55. Grogan PM, Gronseth GS. Practice parameter: steroids, acyclovir, and surgery for Bell's palsy (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2001;56(7):830–836. 10.1212/WNL.56.7.830.
56. Gronseth GS, Paduga R; American Academy of Neurology. Evidence-based guideline update: steroids and antivirals for Bell palsy: report of the Guideline Development Subcommittee of the American Academy of Neurology. *Neurology* 2012;79(22):2209–2213. doi:10.1212/WNL.0b013e318275978c.
57. Sullivan FM, Swan IR, Donnan PT, et al. Early treatment with prednisone or acyclovir in Bell's palsy. *N Engl J Med* 2007;357(16):1598–1607. doi:10.1056/NEJMoa072006.
58. Engström M, Berg T, Stjernquist-Desatnik A, et al. Prednisolone and valaciclovir in Bell's palsy: a randomised, double-blind, placebo-controlled, multicentre trial. *Lancet Neurol* 2008;7(11):993–1000. doi:10.1016/S1474-4422(08)70221-7.
59. Tan ST, Staiano JJ, Itinteang T, et al. Gold weight implantation and lateral tarsorrhaphy for upper eyelid paralysis. *J Craniomaxillofac Surg* 2013;41(3):e49–e53. doi:10.1016/j.jcms.2012.07.015.
60. Reich SG. Bell palsy: which side? *Neurology* 2007;68(2):E1. doi:10.1212/01.wnl.0000250353.75532.29.
61. Yalthro TC, Jankovic J. The many faces of hemifacial spasm: differential diagnosis of unilateral facial spasms. *Mov Disord* 2011;26(9):1582–1592. doi:10.1002/mds.23692.
62. Reich SG, Grill SE. Gustatory sweating: Frey syndrome. *Neurology* 2005;65(11):E24. doi:10.1212/01.wnl.0000182298.85958.46.