



## Bell's Palsy: Nursing Management, Interventions, And Implications for Patient Care and Recovery-An Updated Review Article

<sup>1</sup>-Hassan Ganem Mohammed Haqawi,<sup>2</sup>-Mostafa Ahmed Ibrahim Sabei,<sup>3</sup>-Mohsen Ali Mustafa Almuhammad, <sup>4</sup>-Fahad Ali Mosa Dallak,<sup>5</sup>-Mohamed Ali Mohammed Shamaki, <sup>6</sup>-Ahlam Ibrahim Hassan Haqawi,<sup>7</sup>- Salha Ali Mansour Shamakhi,<sup>8</sup>-Yahia Mohamed Ibraheem Khoderi,<sup>9</sup>- Laheg Ahmad Ali Dallak,<sup>10</sup>- Hind Hamoud Khader Kherat

<sup>1</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>2</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>3</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>4</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>5</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>6</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>7</sup> Ksa, Ministry Of Health

<sup>8</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>9</sup> Ksa, Ministry Of Health, Baish General Hospital

<sup>10</sup> Ksa, Ministry Of Health, Baish General Hospital

### Abstract:

**Background:** Bell's palsy is a common cause of peripheral facial paralysis, accounting for 60-75% of facial paralysis cases. The condition is of unknown etiology, though it is often linked to viral infections, particularly herpes simplex virus (HSV) and herpes zoster virus (HZV). Risk factors such as diabetes, hypertension, and pregnancy complications can exacerbate its onset. The facial nerve's anatomy and its involvement in facial expression, sensation, and parasympathetic functions are central to understanding the condition's pathophysiology. Symptoms of Bell's palsy include facial drooping, pain, altered taste, and hearing sensitivity, with diagnosis based largely on clinical evaluation and exclusion of other causes.

**Aim:** The review aims to analyze current research on Bell's palsy, its management, nursing interventions, and implications for patient care and recovery.

**Methods:** A narrative review was conducted by analyzing studies published between 2011 and 2022 from databases such as PubMed, Medscape, and the Cochrane Database. The review incorporated articles focused on the pathogenesis, diagnosis, treatment, and nursing management of Bell's palsy, including primary sources and older studies for contextual understanding.

**Results:** The review highlights the importance of early intervention in improving recovery outcomes. Corticosteroids, often combined with antivirals, are typically used in treatment. Early recovery within three weeks is a strong predictor of full recovery. Despite treatment, some patients experience complications, including recurrence, and about 4-14% face recurrence of symptoms. The facial nerve's anatomical vulnerability and the effects of viral reactivation contribute significantly to the development and severity of the disease.

**Conclusion:** Bell's palsy remains a challenging condition with an uncertain cause. Timely diagnosis and treatment, particularly corticosteroids, enhance the likelihood of full recovery. Further research into optimal treatment regimens and nursing strategies is necessary to improve patient outcomes.

**Key Words:** Bell's Palsy, Facial Paralysis, Corticosteroids, Nursing Management, Recovery, Herpes Simplex Virus, Herpes Zoster Virus.

Received: 05 October 2024

Revised: 19 November 2024

Accepted: 02 December 2024

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## **Introduction:**

The human face is a critical aspect of an individual's identity and distinctiveness, with facial expressions playing a pivotal role in conveying emotions and facilitating social interactions. Consequently, any impairment in the control of facial muscles, alongside physical disabilities, leads to both social and psychological distress [1]. Bell's palsy is a form of acute-onset peripheral facial neuropathy, serving as one of the leading causes of lower motor neuron facial paralysis. This condition accounts for approximately 60-75% of all facial paralysis cases. Annually, 7-40 cases occur per 100,000 individuals, with prevalence rates being approximately equal among both genders. Although the precise cause remains idiopathic, Bell's palsy has a notable association with specific viral infections, leading to inflammation of the nerve, which in turn causes focal edema, demyelination, and ischemia. According to various studies, several risk factors, including elevated blood glucose levels [2], uncontrolled hypertension, severe pre-eclampsia [3], migraines [4], and exposure to radiation [5], exacerbate the pathophysiological mechanisms, increasing susceptibility to the condition. The degree of weakness may be either complete or partial, often accompanied by symptoms such as numbness, mild pain, heightened sensitivity to sound, and changes in taste sensation. The diagnosis is largely one of exclusion and is primarily based on clinical examination.

An understanding of the neuroanatomy of the facial nerve can be instrumental in distinguishing between central and peripheral lesions, a critical differentiation since the management strategies vary according to the etiology. Given the questionable efficacy of antiviral medications, they are generally recommended in conjunction with corticosteroids. Patients who exhibit signs of recovery within the first three weeks after symptom onset have a significantly higher likelihood of achieving full recovery. Thus, earlier intervention reduces the risk of complications and residual paraesthesias. Approximately 4-14% of patients may experience recurrence, with 36% of cases occurring on the same side [6]. This narrative review systematically analyzed the existing literature on Bell's palsy, utilizing multiple databases, including PubMed, WebMD, Medscape, Google Scholar, and the Cochrane Database of Systematic Reviews, as well as standard textbooks. An advanced search was conducted on these platforms, with articles published between 2011 and 2022 being included for review. The following search terms were used in various combinations: 'Bell's palsy,' 'facial palsy,' 'Bell's phenomenon,' and 'idiopathic facial paralysis.' The reference lists of pertinent publications were also examined as potential sources of further relevant information. Older studies, specifically those over ten years old, were incorporated into the discussion section when deemed essential for the comprehensiveness of this review. However, no efforts were made to uncover unpublished data.

Bell's palsy is named after Sir Charles Bell (1774-1842), who was the first to present the anatomical basis of the condition. However, recent research has acknowledged the contributions of other European medical practitioners who made earlier clinical observations and documented cases of seventh peripheral cranial nerve palsy [7]. The first reported case of idiopathic facial paralysis is attributed to the 18th-century professor of medicine, Nicolaus Friedrich, in Wurzburg. This case report described three middle-aged men who experienced episodes of unilateral facial paralysis, which were either subacute or acute in nature, with gradual improvement over weeks or months. Later, Charles Bell conducted studies on facial nerve function in animals, and during his surgical practice in London, he encountered several cases of unilateral facial nerve paralysis. His most famous and frequently cited case, published in 1828, involved a man who was struck by a bull, resulting in permanent facial nerve paralysis [8].

## **Anatomical Perspective**

A thorough understanding of the etiopathogenesis of Bell's palsy necessitates a foundational knowledge of the facial nerve's course and innervation. The facial nerve is comprised of three nuclei: the motor, sensory, and parasympathetic nuclei. The course of the facial nerve can be divided into six distinct

segments. The first segment, known as the intracranial segment, houses the motor nucleus of the facial nerve located in the pons, from which motor fibers originate. These fibers curve around the abducens nerve nucleus and are joined by the intermediate nerve, which carries both sensory and parasympathetic components. This mixed nerve passes through the posterior cranial fossa and enters the bony facial canal (also referred to as the fallopian canal) through the anterior superior quadrant of the internal acoustic meatus. This segment is known as the meatal or canalicular segment. Within the inner ear, the facial nerve traverses the fallopian canal situated between the cochlea and vestibule, before it turns posteriorly at the geniculate ganglion, forming the first genu. This segment is the shortest and narrowest, rendering it particularly susceptible to inflammation and ischemia. It is termed the labyrinthine segment. The labyrinthine segment then extends to form the tympanic segment in the middle ear, takes another turn just distal to the pyramidal eminence (second genu), and descends vertically as the mastoid segment. The bony fallopian canal, which can often be dehiscent in certain areas, is more prone to damage in such locations. The mastoid segment begins at the second genu, gives off its branches, and culminates at the stylomastoid foramen, thus forming the extratemporal segment. The facial nerve then passes between the superficial and deep lobes of the parotid gland, ultimately terminating into five branches at the anterior border of the gland [8,9].

The facial nerve is responsible for providing efferent motor innervation to all facial muscles involved in expression, the stapedius, and the posterior belly of the digastric muscles, in addition to parasympathetic and sensory fibers [10]. These parasympathetic fibers innervate the submandibular and lacrimal glands via the chorda tympani and the greater superficial petrosal nerve, respectively [8]. Consequently, any damage to the facial nerve can lead to paralysis of the aforementioned structures and fibers. The complex and convoluted pathways, along with the nerve's presence within a narrow bony canal, render the seventh cranial nerve more vulnerable to paralysis than any other nerve in the human body [11].

### **Etiopathogenesis**

The precise pathogenesis of Bell's palsy remains idiopathic; however, various immune, ischemic, and hereditary factors have been strongly associated with its etiology. Recent studies suggest that the reactivation of dormant herpes viruses in the geniculate ganglion, followed by migration to the facial nerve, plays a crucial role in the condition's onset [8,12]. The herpes zoster virus (HZV) and herpes simplex virus (HSV), both neurotropic alpha herpes viruses, are the most commonly implicated pathogens in Bell's palsy [13]. These viruses can remain latent within the ganglia for the duration of an individual's life [14]. Among these, HZV is considered more aggressive, as it spreads along the nerve via satellite cells. Herpes simplex is typically associated with cold sores and genital herpes, whereas herpes zoster causes chickenpox and shingles. A virus is considered latent when no active replication occurs; however, in states of immune deficiency or the presence of antibodies, the virus can cause nerve damage and inflammation, leading to further compression of the facial nerve within its narrow bony canal. Other viruses implicated in Bell's palsy include Epstein-Barr virus, which causes infectious mononucleosis, as well as cytomegalovirus, adenovirus, mumps virus, and influenza B, among others [15]. Vascular ischemia can be categorized as primary, secondary, or tertiary. Primary ischemic neuropathy, which results in inflammation of the affected nerve, is more likely to occur under specific clinical conditions such as diabetes mellitus [10]. It is often triggered by cold exposure or emotional stress. Although the facial nerve has an adequate blood supply and a robust epineurium, vasospasms can reduce blood flow, leading to acute inflammation and primary ischemic neuritis, which is relatively rare [16]. Secondary ischemia may follow, exacerbating nerve damage through increased capillary permeability, fluid accumulation, edema, and nerve compression [17]. A hereditary predisposition in 4-14% of individuals can also narrow the fallopian canal, making the nerve more vulnerable to early compression, even in the presence of minimal edema. This genetic predisposition is usually autosomal dominant and further increases the risk of nerve compression [18].

### **Clinical Presentation**

The onset of symptoms in Bell's palsy is sudden, with severity reaching its peak within 48 to 72 hours. The severity of symptoms can vary, ranging from mild fatigue to severe paralysis of the facial muscles

on the ipsilateral side. Common manifestations of Bell's palsy include the inability to blink or close the eye, the inability to wrinkle the lips or raise the mouth corner, and visible signs such as facial drooping, sagging of the ipsilateral eyebrow, flattening of the nasolabial fold, pain around the ear on the affected side, hearing impairment, and dryness of the eye or mouth [19].

### **Other Symptoms**

Additional symptoms often associated with Bell's palsy include hyperacusis, which arises due to the degeneration of nerve fibers in the stapedius muscle, as well as alterations in taste sensation and dry eyes, both of which are the result of parasympathetic involvement. Some individuals may also experience facial paresthesia, which is frequently mistaken as sensory disturbance but is primarily a manifestation of motor dysfunction. Furthermore, patients may report sensory or hearing loss during their clinical presentation [20].

### **Clinical Examination and Diagnosis**

A comprehensive neurological and general examination is essential in the clinical assessment of Bell's palsy, incorporating evaluations of the ear, ophthalmologic examination, and inspection of the skin and parotid gland [21]. In cases where herpes zoster is suspected, the presence of blisters or scabbing around the ear may suggest Ramsay-Hunt syndrome, which can lead to hearing impairment and facial nerve palsy. Observing the patient during the interview may also uncover subtle manifestations of weakness, offering valuable diagnostic clues [7].

A systematic evaluation approach is critical for accurate diagnosis. The clinician should observe facial expressions and assess facial movements, including the ability to wrinkle the forehead (temporal branch), close the eyes tightly, puff the cheeks (mandibular branch), assess smile symmetry, and observe eye closure and nasal wrinkling (zygomatic and buccal branches). The severity and prognosis of facial nerve palsy can be measured using the House-Brackmann grading system, which categorizes the condition into six grades. Grade 1 indicates normal facial function, whereas Grade 6 represents complete paralysis [22]. In the clinical examination, the absence or asymmetry of forehead wrinkling when raising the eyebrows on the affected side is notable. Additionally, the involved eye may exhibit partial closure, remaining slightly open during attempts to close the eye fully, and the eyelid may demonstrate mild lag during blinking. Bell's phenomenon can be elicited by attempting to open the patient's eyelids while they are asked to tightly close their eyes, during which the eyes deviate upward and laterally. This test also serves to assess the strength of the orbicularis oculi muscle. A thorough examination of the ear is necessary to rule out conditions such as cholesteatoma, acute or chronic suppurative otitis media, malignant otitis media, and other middle ear diseases. A red chorda tympani (vascular flaring of the tympanometry area) is often observed in Bell's palsy. Audiometric tests, including pure tone audiometry, speech audiometry, brainstem evoked response audiometry, and other specialized tests, can help exclude cochlear and retrocochlear lesions.

To determine the affected segment of the facial nerve, clinicians should perform prognostic tests, including evaluations of tearing, salivation, taste, and stapedial reflex, with electrodiagnostic testing providing additional insights into the extent of nerve damage. The diagnosis of Bell's palsy is primarily clinical, involving the exclusion of other potential causes of unilateral facial paralysis [23]. Electrodiagnostic tests, when performed within 14 days of symptom onset, can offer valuable prognostic information. Other conditions, such as central lesions (e.g., stroke or demyelinating diseases), cholesteatoma, parotid gland tumors, middle ear infections, Lyme disease, diabetes, granulomatous disease, Ramsay-Hunt syndrome, trauma, and Guillain-Barré syndrome, can mimic Bell's palsy and must be considered in the differential diagnosis [20,24,25]. A meticulous patient history, comprehensive head and neck examination, and otological assessment are crucial, complemented by radiologic studies and laboratory tests, including peripheral smears, blood counts, blood glucose, sedimentation rates, and serology. Determining whether the forehead muscle is involved in paralysis is key to differentiating between central and peripheral causes of facial nerve palsy. The upper part of the facial nerve nucleus, which innervates the frontalis muscle, receives input from both cerebral hemispheres. In contrast, the lower part of the nucleus, which innervates the lower and middle face, is supplied by crossed fibers from a single hemisphere. Therefore, if the lower

face is paralyzed but the forehead function remains intact, the lesion is likely supranuclear. Common signs of peripheral facial nerve palsy include absence of forehead wrinkles, incomplete eyelid closure, sagging eyebrows, flattened nasolabial folds, and drooping of the mouth corner.

Nerve excitability tests are routinely employed to monitor nerve degeneration. These tests record the minimal electrical stimulus required to elicit visible muscle contraction, helping to determine the excitation threshold. By comparing the excitation threshold on the affected side with that of the unaffected side, a significant difference of more than 3.5 mA suggests a poor prognosis. Imaging and laboratory studies become particularly important in cases where symptoms do not improve after three weeks of treatment or in instances of recurrence [8]. MRI enhancement of the facial nerve, when correlated with clinical findings, can confirm the diagnosis of Bell's palsy. While enhancement in the tympanic and vertical segments may be observed in normal individuals, enhancement in the labyrinthine segment is characteristic of Bell's palsy [26]. Motor nerve conduction studies and electromyography of the facial nerve play a pivotal role in guiding the pharmacotherapeutic and surgical management of the patient. These electrodiagnostic tests can measure the action potentials in the muscles involved, helping to assess the extent of axonal damage. Patients exhibiting axonal degeneration greater than 90 percent may benefit from surgical decompression, whereas those with less severe degeneration are typically managed conservatively and tend to have a more favorable prognosis.

### **Overview of Data:**

Bell's palsy is characterized as the most prevalent form of peripheral paralysis affecting the seventh cranial nerve, typically presenting with rapid and unilateral onset. It is primarily diagnosed through physical examination as it is considered a diagnosis of exclusion. The facial nerve, which is responsible for both motor and parasympathetic functions, has an intricate path through the intracranial, intratemporal, and extratemporal regions. Its motor function controls facial muscles, particularly those of the upper and lower face, while the parasympathetic component manages the lacrimal and salivary glands, as well as taste sensation in the anterior two-thirds of the tongue. Given the motor role of the facial nerve, particular emphasis is placed on assessing the strength of the forehead muscles when diagnosing Bell's palsy. Preservation of forehead strength suggests a central cause of weakness rather than a peripheral one. The role of antiviral treatments in Bell's palsy has been debated, though most clinical guidelines advocate for a medical approach, typically combining corticosteroids with antiviral therapy to manage the condition effectively [27][28][29].

### **Causes of Bell's Palsy**

Bell's palsy is predominantly idiopathic, though emerging evidence has identified various clinical conditions and pathologies that can manifest with unilateral facial paralysis. Among the most frequently implicated are viral infections, including herpes simplex virus, varicella-zoster virus, and Epstein-Barr virus. In clinical practice, misdiagnosis can occur when Bell's palsy is incorrectly attributed to known etiologies, such as Ramsay-Hunt syndrome or Lyme disease, where facial paralysis is a prominent feature. Despite the variety of potential causes—spanning from idiopathic origins to traumatic, neoplastic, congenital, and autoimmune conditions—approximately 70% of facial nerve palsies are ultimately diagnosed as Bell's palsy. It is important to recognize that while the condition is classified as idiopathic, other identifiable causes may contribute to the presentation of facial paralysis, necessitating careful differential diagnosis to avoid misclassification [30].

### **Risk Factors for Bell's Palsy**

The annual incidence of Bell's palsy is approximately 15 to 20 per 100,000 people, with around 40,000 new cases reported annually. The lifetime risk of developing the condition is estimated at 1 in 60. Although the recurrence rate for Bell's palsy ranges from 8% to 12%, a significant proportion of patients—around 70%—experience complete resolution even without medical intervention. Bell's palsy does not exhibit a gender or racial preference, and it can affect individuals of any age. However, it is most commonly diagnosed in mid to late adulthood, with the median age of onset being 40 years. Several risk factors have

been identified that may predispose individuals to Bell's palsy, including diabetes, pregnancy, preeclampsia, obesity, and hypertension. These factors, while not causal in all cases, are often observed in individuals who develop facial nerve paralysis and may influence the course of the condition [31].

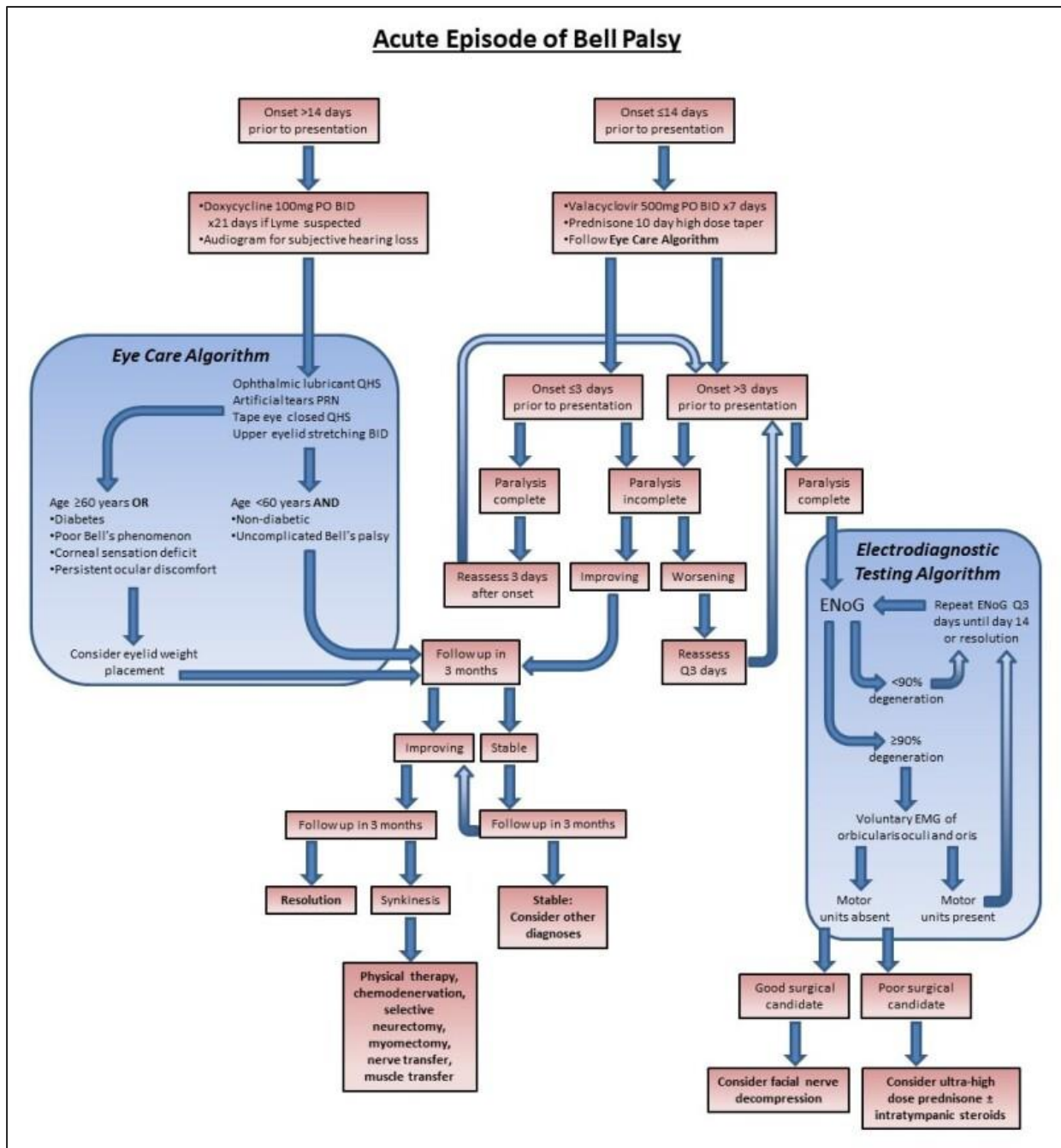


Figure 1: Acute Episodes of Bell's Palsy.

### Nursing Diagnosis for Bell's Palsy

The nursing diagnosis for Bell's palsy focuses on the management and relief of symptoms associated with the condition, which primarily involves facial nerve dysfunction. Key symptoms include facial weakness or paralysis, which can impact a patient's ability to perform essential activities such as closing the eyelids or smiling. Consequently, a primary concern is the prevention of injury, especially due to the inability to blink or protect the affected eye, which can lead to dryness, irritation, or potential corneal damage. Nurses should also address emotional and psychological challenges, as the visible facial asymmetry may affect a patient's self-esteem and social interactions. The loss of taste sensation, along with

possible difficulties in eating or drinking, should be assessed to ensure adequate nutrition and hydration. Additionally, the nursing diagnosis may include addressing impaired social interaction due to altered facial appearance or communication difficulties. Nurses play a crucial role in educating patients and caregivers about the nature of the condition, the expected course of recovery, and strategies to manage symptoms, including the use of lubricating eye drops, facial exercises, and protective measures for the affected eye. The ultimate goal of nursing care is to alleviate discomfort, promote recovery, and support the patient's emotional well-being while facilitating optimal functional recovery from Bell's palsy [31].

### **Assessment of Bell's Palsy**

Patients with Bell's palsy typically present rapid and progressive symptoms that manifest over a period of one day to one week, often reaching their peak severity within 72 hours. The weakness, which may be partial or complete, typically affects one side of the face, leading to a noticeable reduction in strength around the eyebrows, forehead, and corner of the mouth. One of the hallmark signs of the condition is the inability to fully close the eyelid or lip on the affected side. During the physical examination, particular attention should be given to the forehead muscles. If there is preserved strength in the forehead, the possibility of a central cause, such as a stroke, should be considered. Patients may also report additional symptoms, such as altered taste, hypersensitivity to sound, and changes in tearing or salivation. These accompanying symptoms may help clinicians differentiate Bell's palsy from other causes of facial paralysis and guide further diagnostic evaluation [32-33].

### **Evaluation**

The evaluation of Bell's palsy primarily involves a comprehensive history and physical examination. A critical component of this process is the use of the House-Brackmann Facial Nerve Grading System, which allows clinicians to assess the extent of facial nerve weakness. This system categorizes weakness on a scale from grade I (no weakness) to grade VI (complete paralysis). In cases where Bell's palsy is suspected, laboratory or radiographic tests are not typically required unless atypical features are present, in which case further investigation for a central cause of the symptoms may be warranted. For instance, Lyme disease testing is considered when there is a history of potential tick exposure, including a prior tick bite, skin rash, or arthritis. Routine Lyme disease testing is not advised in the absence of these symptoms. Additionally, diabetic testing is unnecessary, as facial nerve palsy is not classified as diabetic neuropathy. The timing of imaging for suspected Lyme disease is debated, though the majority of experts suggest performing an MRI if facial palsy persists after two months without improvement. MRI is the preferred imaging modality as it can identify inflammation of the facial nerve, along with ruling out other potential causes such as schwannoma, hemangioma, or space-occupying lesions [32].

### **Medical Management**

Corticosteroids represent the cornerstone of treatment for Bell's palsy, with typical regimens recommending doses of 60 mg to 80 mg daily for approximately one week. Evidence suggests that the combination of corticosteroids and antivirals may offer improved outcomes compared to corticosteroids alone. A 2009 meta-analysis concluded that the addition of antivirals did not achieve statistical significance in enhancing the outcome when compared to corticosteroids alone. For patients presenting with severe facial nerve palsy (House-Brackmann grade IV or greater), combination therapy involving both corticosteroids and antivirals may be offered. However, no significant increase in adverse reactions has been observed with antivirals when compared to placebo or corticosteroids. Patients should also be advised to apply eye lubrication and use a patch to protect the affected eye at night, reducing the risk of corneal abrasions. Surgical interventions may be considered for those with persistent symptoms after several weeks or months, with options ranging from eyelid weights to muscle transfers. Despite its potential, facial nerve decompression surgery has not been proven to be a universally effective treatment and is generally reserved for specific cases, given the poor quality of prior studies in this area. For patients who fail to improve within four weeks, early referral to a specialist (such as plastic surgery, neurology, or otolaryngology) is recommended for more aggressive treatment options [33][34][35].

## **Nursing Management**

Effective nursing management for patients with Bell's palsy involves several key strategies aimed at promoting recovery and preventing complications. One critical component is ensuring that the affected eye remains lubricated to prevent dryness and irritation, which can lead to corneal damage. Additionally, patients should be instructed to wear a patch at night to protect the eye from accidental injury or scratching. Regular follow-up with an eye doctor is necessary to monitor eye health, particularly if the patient experiences difficulties with eye closure. When outside, wearing sunglasses can protect the affected eye from environmental irritants such as wind and dust. To address facial muscle weakness, nurses should encourage patients to perform facial muscle exercises regularly to maintain muscle tone and function. It is also important for patients to modify their eating habits, such as eating on the opposite side of the mouth to avoid difficulty with chewing or swallowing. Regular dental visits are advised to ensure oral health is maintained, as the inability to fully close the mouth may increase the risk of oral infections. Nurses play an essential role in educating patients and caregivers about these interventions and the importance of timely medical follow-up for optimal management of Bell's palsy [34].

## **When To Seek Help**

Patients with Bell's palsy should seek medical attention promptly if they experience worsening symptoms, particularly if the affected eye becomes red, painful, or if there is any loss of vision. These symptoms may indicate complications, such as corneal damage, which requires immediate medical intervention to prevent further deterioration of vision or additional injury to the eye.

## **Outcome Identification**

The primary goal for patients with Bell's palsy is the restoration of normal facial muscle function and facial sensation. Successful outcomes are typically characterized by the complete recovery of facial movement, including the ability to raise the eyebrows, close the eyes fully, and smile symmetrically. Additionally, the resolution of taste abnormalities and the restoration of normal salivation are important indicators of recovery. Achieving these outcomes is critical to improving the patient's quality of life and reducing the psychological impact of facial paralysis.

## **Monitoring**

Ongoing monitoring is essential in managing Bell's palsy and ensuring recovery. Key aspects of monitoring include checking the patient's vision to detect any signs of eye dryness or corneal injury, assessing neurovital signs to track changes in neurological status, and determining the patient's ability to close the affected eyelid fully. Regular evaluations help guide treatment adjustments and prevent complications such as eye damage or permanent facial muscle weakness.

## **Coordination of Care**

Bell's palsy is the most prevalent cause of unilateral facial paralysis. Despite being a benign condition, it carries moderate morbidity that can significantly impact the quality of life. The etiology of Bell's palsy remains unknown, and the treatment approach continues to be a topic of debate within the medical community. Although corticosteroids and/or antiviral agents are commonly prescribed, there are no randomized controlled trials to definitively determine which of these treatments is more effective. Compounding this issue is the fact that a substantial number of cases resolve without intervention. However, for individuals who experience prolonged facial paralysis, particularly with poor speech, incomplete eyelid closure, or aesthetic concerns, intervention is necessary and should involve a multidisciplinary team. Given that Bell's palsy affects multiple organ systems, a collaborative approach, involving various healthcare professionals, has been shown to yield better outcomes. The central focus of treatment should prioritize the individual's overall well-being rather than merely addressing symptoms. The advent of botulinum toxin has significantly alleviated the long-term effects of this condition. Surgical options are considered a last resort, particularly in chronic cases. Fortunately, facial muscles can remain viable for several years, allowing for potential complex reconstructive surgery. However, rather than

immediately resorting to invasive procedures with no guaranteed outcomes, early recognition and timely initiation of steroid therapy are recommended as the most effective approach [36].

### **Outcomes**

There is a notable lack of robust evidence-based guidelines regarding the treatment and outcomes of Bell's palsy. The absence of comprehensive clinical trials is exacerbated by the fact that a majority of cases resolve spontaneously over time. Consequently, most of the available outcome data are derived from case reports or small-scale case series. While most patients eventually experience recovery, the process can take several months or, in some instances, years for full recovery. Due to the range of available treatment options, including pharmacologic and non-pharmacologic interventions, a multidisciplinary team approach is essential to tailor treatment to individual needs, as patients often exhibit varied responses to different therapies. As the course of Bell's palsy is unpredictable, treatment outcomes should be continually monitored, and interventions adjusted accordingly to optimize recovery [37].

### **Health Teaching and Health Promotion**

Health education plays a critical role in the management of Bell's palsy. Patients should be instructed to maintain regular eye lubrication to prevent dryness and potential damage, which is especially important given the difficulty some patients experience in closing the affected eyelid. Additionally, wearing a patch at night can help protect the eye from injury, particularly when the eyelid cannot fully close. Regular visits to an eye care specialist are essential to monitor and address any ocular complications. To protect the affected eye from environmental irritants, wearing sunglasses when outside is recommended. Patients should also engage in facial muscle exercises to promote recovery and reduce the risk of long-term facial weakness. It is also advised that patients modify their eating habits, such as eating on the unaffected side of the mouth to avoid difficulty while chewing. Regular dental visits are essential to prevent complications that may arise from difficulty closing the mouth. These measures collectively support the patient's recovery process and contribute to minimizing the long-term impact of Bell's palsy.

### **Risk Management**

Whenever a patient experiences visual impairment, it is essential that they are promptly referred to an ophthalmologist to ensure proper evaluation and management. This is critical, as Bell's palsy, despite being primarily a condition affecting the facial nerve, can lead to significant visual complications when eye protection and lubrication are not adequately addressed. Ensuring that eye dryness is prevented through the use of artificial tears or other eye lubrication solutions is a key aspect of managing the patient's overall well-being during recovery from Bell's palsy. Proper management and early intervention by an ophthalmologist are crucial in preventing further vision-related complications, which can arise from incomplete eyelid closure, a common issue in patients with this condition. Additionally, educating the patient and their healthcare team about the importance of regular eye care, including the use of appropriate ocular lubricants and other protective measures, will significantly reduce the risk of permanent visual impairment. Early referral and consistent management ensure that any potential eye complications are addressed in a timely manner, contributing to better long-term outcomes for patients suffering from Bell's palsy [38].

### **Discharge Planning**

Bell's palsy remains the most common cause of unilateral facial paralysis. Although it is generally considered a benign condition, it is associated with moderate morbidity, which can significantly affect the patient's quality of life and, in some cases, lead to vision loss. Given these potential complications, the condition is best managed by a multidisciplinary team of healthcare professionals. The underlying etiology of Bell's palsy remains elusive, and the management approach continues to be a subject of clinical debate. While corticosteroids and/or antiviral medications are often prescribed as first-line treatments, the lack of randomized controlled trials makes it difficult to determine the optimal therapeutic approach. The challenge is further exacerbated by the fact that a significant proportion of Bell's palsy cases resolve spontaneously. Nonetheless, for patients with persistent facial paralysis, especially those experiencing

difficulties such as speech impairment, incomplete eyelid closure, or aesthetic concerns, treatment should be tailored by a collaborative team. This team approach is especially necessary because the disorder involves multiple organ systems, making it essential to provide comprehensive care. It is vital that the treatment plan focuses on the individual patient's needs and well-being rather than merely addressing the symptoms. In addition to the primary clinicians, all members of the healthcare team, including pharmacists and nurse practitioners, must emphasize the importance of eye protection and lubrication. Preventing eye dryness through the use of lubricating solutions is a critical aspect of the patient's care plan [38].

### **Other Issues**

Misdiagnosis of Bell's palsy occurs in a significant proportion of cases, with an estimated rate of up to 10.8%. Therefore, obtaining a thorough history and conducting a detailed physical examination are crucial in accurately diagnosing the condition. A particular focus should be placed on assessing the forehead muscles, as Bell's palsy, a peripheral facial nerve palsy, invariably involves the muscles of the forehead. The history and physical exam provide guidance for further testing to determine the underlying cause of facial nerve weakness. Routine testing for Lyme disease is not recommended for all patients, but rather, only for those who present with a history of tick bites or have accompanying symptoms such as a skin rash or arthritis. Many patients with Bell's palsy can be treated at home with appropriate medical management and close follow-up to monitor for symptom improvement. However, if there is little to no improvement within the initial weeks, referral to a specialist should be considered to explore further treatment options. Although there are no known preventative measures for Bell's palsy, it is important to note that 8% to 12% of patients may experience a recurrence of the condition. Therefore, regular follow-up and monitoring are essential to ensure optimal care and address any potential recurrence or complications in a timely manner [38].

### **Conclusion:**

Bell's palsy is a complex neurological disorder that results in sudden facial paralysis, primarily affecting the motor function of the facial nerve. It is typically idiopathic, although viral reactivation—especially of herpes simplex and herpes zoster viruses—plays a significant role in its onset. The condition's diagnosis is clinical, with exclusion of other causes of unilateral facial paralysis such as stroke, Lyme disease, or Ramsay-Hunt syndrome. The condition's severity can range from mild facial weakness to complete paralysis, with associated symptoms like pain, altered taste, and hyperacusis. Clinical examination is essential to diagnose Bell's palsy and differentiate it from central nervous system lesions or other pathologies. The early intervention, primarily with corticosteroids, is crucial in preventing long-term complications. Although antivirals are often used alongside steroids, their efficacy remains uncertain. Recovery rates are generally favorable, with patients showing significant improvement if they begin to recover within three weeks of symptom onset. However, recurrence is not uncommon, and a small proportion of patients may experience incomplete recovery or persistent symptoms such as facial weakness or sensory disturbances. Nurses play a vital role in the management of Bell's palsy, particularly in providing care during the acute phase and educating patients about symptom monitoring and recovery expectations. The use of the House-Brackmann grading system is helpful in assessing the severity of the condition and predicting recovery outcomes. In addition to pharmacological treatments, supportive care interventions are necessary to address the emotional and psychological impact of facial paralysis. Patients often experience significant distress due to changes in appearance and difficulties with communication, necessitating psychological support and counseling. Given the idiopathic nature of Bell's palsy, further research is required to better understand its pathophysiology and optimize treatment strategies. Nursing interventions focused on prevention, early intervention, and comprehensive patient care are essential to improving recovery outcomes and reducing the risk of long-term complications. Further clinical trials are needed to explore the most effective treatment combinations and the role of nursing care in facilitating recovery.

## References:

1. Efficacy of laser acupuncture for patients with chronic Bell's palsy: a study protocol for a randomized, double-blind, sham-controlled pilot trial. Ton G, Lee LW, Ng HP, et al. *Medicine (Baltimore)* 2019;98:0. doi: 10.1097/MD.00000000000015120.
2. Bell's palsy: a manifestation of prediabetes? Bosco D, Plastino M, Bosco F, et al. *Acta Neurol Scand.* 2011;123:68–72. doi: 10.1111/j.1600-0404.2010.01365.x.
3. LMN facial palsy in pregnancy: an opportunity to predict preeclampsia-report and review. Aditya V. *Case Rep Obstet Gynecol.* 2014;2014:626871. doi: 10.1155/2014/626871.
4. Increased risk of Bell palsy in patients with migraine: a nationwide cohort study. Peng KP, Chen YT, Fuh JL, Tang CH, Wang SJ. *Neurology.* 2015;84:116–124. doi: 10.1212/WNL.0000000000001124
5. Radiation exposure and Bell's palsy: a hypothetical association. Khateri M, Cheraghi S, Ghadimi A, Abdollahi H. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6169125/#:~:text=Radiation%2Dinduced%20Bell's%20palsy%20has,been%20exposed%20to%20UV%20radiation>. *J Biomed Phys Eng.* 2018;8:337–340.
6. says RK. Bell's Palsy Recovery. [ Aug; 2021 ]. 2009. <https://www.news-medical.net/health/Bells-Palsy-Recovery.aspx> <https://www.news-medical.net/health/Bells-Palsy-Recovery.aspx>
7. The history of facial palsy and spasm: Hippocrates to Razi. Sajadi MM, Sajadi MR, Tabatabaie SM. *Neurology.* 2011;77:174–178. doi: 10.1212/WNL.0b013e3182242d23.
8. The neurologist's dilemma: a comprehensive clinical review of Bell's palsy, with emphasis on current management trends. Zandian A, Osiro S, Hudson R, Ali IM, Matusz P, Tubbs SR, Loukas M. *Med Sci Monit.* 2014;20:83–90. doi: 10.12659/MSM.889876.
9. Gaillard F. Facial nerve. [ Jul; 2022 ]. 2022. <https://radiopaedia.org/articles/facial-nerve> <https://radiopaedia.org/articles/facial-nerve>
10. The etiology of Bell's palsy: a review. Zhang W, Xu L, Luo T, Wu F, Zhao B, Li X. *J Neurol.* 2020;267:1896–1905. doi: 10.1007/s00415-019-09282-4.
11. Histopathological changes in the temporal bone in Bell's palsy. Michaels L. *Acta Otolaryngol Suppl.* 1990;470:114–117. doi: 10.3109/00016488909138364.
12. Bell's palsy: aetiology, clinical features and multidisciplinary care. Eviston TJ, Crosson GR, Kennedy PG, Hadlock T, Krishnan AV. *J Neurol Neurosurg Psychiatry.* 2015;86:1356–1361. doi: 10.1136/jnnp-2014-309563.
13. Global and regional estimates of prevalent and incident herpes simplex virus Type 1 infections in 2012. Looker KJ, Magaret AS, May MT, Turner KM, Vickerman P, Gottlieb SL, Newman LM. *PLoS One.* 2015;10:0. doi: 10.1371/journal.pone.0140765.
14. Bell's palsy - Symptoms and causes. [ Jul; 2022 ]. <https://www.mayoclinic.org/diseases-conditions/bells-palsy/symptoms-causes/syc-20370028>. <https://www.mayoclinic.org/diseases-conditions/bells-palsy/symptoms-causes/syc-20370028>.
15. A comparison of herpes simplex virus type 1 and varicella-zoster virus latency and reactivation. Kennedy PG, Rovnak J, Badani H, Cohrs RJ. *J Gen Virol.* 2015;96:1581–1602. doi: 10.1099/vir.0.000128.
16. Facial palsy, a disorder belonging to influential neurological dynasty: review of literature. Newadkar UR, Chaudhari L, Khalekar YK. *N Am J Med Sci.* 2016;8:263–267. doi: 10.4103/1947-2714.187130.

17. Bell's palsy-tertiary ischemia: an etiological factor in residual facial palsy. Grewal DS. *Indian J Otolaryngol Head Neck Surg.* 2018;70:374–379. doi: 10.1007/s12070-018-1381-9.
18. A meta-analysis uncovers the first sequence variant conferring risk of Bell's palsy. Skuladottir AT, Bjornsdottir G, Thorleifsson G, et al. *Sci Rep.* 2021;11:4188. doi: 10.1038/s41598-021-82736-w.
19. Everything about Bell's Palsy. [ Jul; 2022 ]. 2018. <https://dentagama.com/news/bells-palsy-unilateral-facial-paralysis> <https://dentagama.com/news/bells-palsy-unilateral-facial-paralysis>
20. Bell palsy: Clinical examination and management. Patel DK, Levin KH. *Cleve Clin J Med.* 2015;82:419–426. doi: 10.3949/ccjm.82a.14101.
21. Neuro-ophthalmological approach to facial nerve palsy. Portelinha J, Passarinho MP, Costa JM. *Saudi J Ophthalmol.* 2015;29:39–47. doi: 10.1016/j.sjopt.2014.09.009.
22. sorenсенclinic.com: Jesper Sorensen MD PhD. House Brackmann. *Plastic and Reconstructive Surgeon.* [ Jul; 2022 ]. <https://sorensenclinic.com/microsurgery/house-brackmann/> <https://sorensenclinic.com/microsurgery/house-brackmann/>
23. Surgical interventions for the early management of Bell's palsy. McAllister K, Walker D, Donnan PT, Swan I. *Cochrane Database Syst Rev.* 2013;0. doi: 10.1002/14651858.CD007468.pub3.
24. Etiology, diagnosis, and management of facial palsy: 2000 patients at a facial nerve center. Hohman MH, Hadlock TA. *Laryngoscope.* 2014;124:0–93. doi: 10.1002/lary.24542.
25. The diagnosis and treatment of idiopathic facial paresis (Bell's palsy) Heckmann JG, Urban PP, Pitz S, Guntinas-Lichius O, Gágyor I. *Dtsch Arztebl Int.* 2019;116:692–702. doi: 10.3238/arztebl.2019.0692.
26. Normal and pathological findings for the facial nerve on magnetic resonance imaging. Al-Noury K, Lotfy A. *Clin Radiol.* 2011;66:701–707. doi: 10.1016/j.crad.2011.02.012.
27. Hohman MH, Hadlock TA. Etiology, diagnosis, and management of facial palsy: 2000 patients at a facial nerve center. *Laryngoscope.* 2014 Jul;124(7):E283-93.
28. Escalante DA, Malka RE, Wilson AG, Nygren ZS, Radcliffe KA, Ruhl DS, Vincent AG, Hohman MH. Determining the Prognosis of Bell's Palsy Based on Severity at Presentation and Electroneuronography. *Otolaryngol Head Neck Surg.* 2022 Jan;166(1):151-157.
29. 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.
30. Adour KK, Byl FM, Hilsinger RL, Kahn ZM, Sheldon MI. The true nature of Bell's palsy: analysis of 1,000 consecutive patients. *Laryngoscope.* 1978 May;88(5):787-801.
31. Berkowitz C. DEFINING A DISCOVERY: PRIORITY AND METHODOLOGICAL CONTROVERSY IN EARLY NINETEENTH-CENTURY ANATOMY. *Notes Rec R Soc Lond.* 2014 Dec 20;68(4):357-72.
32. Sullivan FM, Swan IR, Donnan PT, Morrison JM, Smith BH, McKinstry B, Davenport RJ, Vale LD, Clarkson JE, Hammersley V, Hayavi S, McAteer A, Stewart K, Daly F. Early treatment with prednisolone or acyclovir in Bell's palsy. *N Engl J Med.* 2007 Oct 18;357(16):1598-7.
33. Hato N, Yamada H, Kohno H, Matsumoto S, Honda N, Gyo K, Fukuda S, Furuta Y, Ohtani F, Aizawa H, Aoyagi M, Inamura H, Nakashima T, Nakata S, Murakami S, Kiguchi J, Yamano K, Takeda T, Hamada M, Yamakawa K. Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. *Otol Neurotol.* 2007 Apr;28(3):408-13.
34. Jowett N, Hadlock TA. Contemporary management of Bell palsy. *Facial Plast Surg.* 2015 Apr;31(2):93-102.

35. Miller MQ, Hadlock TA. Beyond Botox: Contemporary Management of Nonflaccid Facial Palsy. *Facial Plast Surg Aesthet Med*. 2020 Mar/Apr;22(2):65-70
36. Spencer CR, Irving RM. Causes and management of facial nerve palsy. *Br J Hosp Med (Lond)*. 2016 Dec 02;77(12):686-691.
37. Murakami S, Mizobuchi M, Nakashiro Y, Doi T, Hato N, Yanagihara N. Bell palsy and herpes simplex virus: identification of viral DNA in endoneurial fluid and muscle. *Ann Intern Med*. 1996 Jan 01;124(1 Pt 1):27-30.
38. Warner, M. J., Hutchison, J., Varacallo, M., & Busby, T. H. (2023). Bell Palsy (Nursing). In StatPearls [Internet]. StatPearls Publishing.

شلل بيل: إدارة التمريض، التدخلات، و آثارها على رعاية المريض والتعافي - مقال مراجع محدث

#### الملخص:

الخلفية: يُعد شلل بيل سببًا شائعًا للشلل الوجهي المحيطي، حيث يمثل 60-75% من حالات الشلل الوجهي. الحالة غير معروفة السبب، على الرغم من أنها غالبًا ما ترتبط بالعدوى الفيروسية، خصوصًا فيروس الهربس البسيط (HSV) وفيروس الهربس النطاقي (HZV). يمكن أن تُفاقم عوامل الخطر مثل السكري وارتفاع ضغط الدم ومضاعفات الحمل من ظهورها. تعتبر تشريح العصب الوجهي ومشاركته في التعبير الوجهي والإحساس والوظائف اللاودية من العناصر الأساسية لفهم الفيزيولوجيا المرضية لهذه الحالة. تشمل أعراض شلل بيل ترهل الوجه، الألم، التغير في حاسة التذوق، وحساسية السمع، مع التشخيص الذي يعتمد بشكل أساسي على التقييم السريري واستبعاد الأسباب الأخرى.

الهدف: يهدف هذا المراجعة إلى تحليل الأبحاث الحالية حول شلل بيل، وإدارته، والتدخلات التمريضية، و آثارها على رعاية المريض والتعافي.

الطرق: تم إجراء مراجعة سردية من خلال تحليل الدراسات المنشورة بين عامي 2011 و2022 من قواعد بيانات مثل PubMed و Medscape وقاعدة بيانات Cochrane. شملت المراجعة مقالات تركز على مسببات المرض، والتشخيص، والعلاج، وإدارة التمريض لشلل بيل، بما في ذلك المصادر الأولية والدراسات القديمة لفهم السياق.

النتائج: تبرز المراجعة أهمية التدخل المبكر في تحسين نتائج التعافي. غالبًا ما يُستخدم الكورتيكوستيرويدات، وغالبًا ما يُجمع مع الأدوية المضادة للفيروسات في العلاج. يُعتبر التعافي المبكر خلال ثلاثة أسابيع مؤشرًا قويًا على التعافي الكامل. رغم العلاج، يعاني بعض المرضى من مضاعفات، بما في ذلك التكرار، ويواجه حوالي 14-4% من المرضى تكرار الأعراض. تساهم هشاشة العصب الوجهي من الناحية التشريحية وتأثيرات إعادة تنشيط الفيروس بشكل كبير في تطور وشدة المرض.

الاستنتاج: لا يزال شلل بيل حالة صعبة ذات سبب غير مؤكد. يعزز التشخيص والعلاج في الوقت المناسب، خاصة الكورتيكوستيرويدات، من احتمالية التعافي الكامل. هناك حاجة إلى مزيد من البحث حول أنظمة العلاج المثلى واستراتيجيات التمريض لتحسين نتائج المرضى.

الكلمات الرئيسية: شلل بيل، الشلل الوجهي، الكورتيكوستيرويدات، إدارة التمريض، التعافي، فيروس الهربس البسيط، فيروس الهربس النطاقي.