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Chiari malformation type I and obstructive sleep apnea

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Abstract
Chiari Malformation Type I (CMI) is a structural anomaly of the craniocervical junction, characterized by herniation of the cerebellar tonsils through the foramen magnum. This neural compression results in Sleep-Disordered Breathing (SDB), historically associated with Central Sleep Apnea (CSA). CSAS results from direct dysfunction of the respiratory centers in the brainstem, compromising the neurological drive and causing the brain to fail to send the command to breathe. However, recent evidence highlights a complex prevalence of Obstructive Sleep Apnea (OSA) in patients with CMI. OSA is a disorder of recurrent collapse of the upper airways, which, in this context, is attributed to a secondary mechanism: compression at the craniocervical junction can damage the lower cranial nerves (Glossopharyngeal IX, Vagus X, and Hypoglossal XII). Dysfunction of these nerves results in loss of tone in the dilator muscles of the pharynx and larynx, culminating in airway obstruction and, often, the manifestation of mixed OSA. The need to synthesize the actual prevalence and mechanisms of OSA *versus* CSA is crucial to optimize therapeutic management. Polysomnography (PSG) is the gold standard for this differentiation, as it evaluates respiratory effort to distinguish obstructive from central events. Accurate diagnosis guides treatment, which may begin with surgical decompression of the posterior fossa (DFP). In cases of persistent residual OSA after surgery, adjuvant therapies such as CPAP become essential.
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INTRODUCTION

Chiari Malformation Type I (CMI) is a craniovertebral anomaly characterized by caudal herniation of the cerebellar tonsils through the foramen magnum into the cervical spinal canal. It is the most prevalent subtype of Chiari malformations and can cause a wide variety of neurological symptoms resulting from compression of the brainstem, cerebellum, and medulla oblongata. The clinical relevance of CCHM extends to Sleep-Disordered Breathing (SDB), since bulbar compression can directly alter the respiratory control centers, favoring the occurrence of Central Sleep Apnea (CSA).

Within the spectrum of SUDs, Obstructive Sleep Apnea (OSA) is the most common form, characterized by recurrent episodes of upper airway collapse, leading to hypoxemia and sleep fragmentation. Although OSA is classically associated with oropharyngeal anatomical factors and obesity, recent evidence has demonstrated its significant presence in individuals with MCtI. Tonsillar herniation can compromise the lower cranial nerves—especially the glossopharyngeal and vagus nerves—contributing to reduced pharyngeal tone and predisposing to obstructive or mixed events during sleep.

Recent studies, such as those by Arrell et al. (2024) and Jarrel et al. (2024), highlight the high prevalence of OSA in patients with MCtI and reinforce the need for systematic screening in this population. However, the literature remains heterogeneous regarding the relationship between the anatomy of CTM and the prevalence of OSA, as pointed out by Moore et al. (2022) and Vagianou et al. (2022). Thus, the research problem lies in the need to synthesize the actual prevalence and pathophysiological factors that distinguish OSA from ACS in individuals with CTM.

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This study aims to analyze and integrate evidence on the presence and mechanisms of OSA in TCM, based on the hypothesis that this malformation is associated not only with ACS but also with a significant increase in obstructive events. The scientific and social justification is based on the optimization of diagnosis and therapeutic management. The investigation focuses exclusively on TCMI and OSA, using polysomnography as the gold standard method and evaluating, when applicable, the impact of surgical decompression on the obstructive component.

THEORETICAL FRAMEWORK

This Theoretical Framework establishes the conceptual basis for understanding the complex relationship between Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB), with an emphasis on OSA. Recent literature is used to outline the clinical relevance of the coexistence of these conditions and differentiate the pathophysiology of OSA, which is characterized by mechanical collapse of the upper airway, from the neurological implications resulting from CM-I. The assumption is that these entities, although distinct, can interact significantly, affecting ventilatory control, craniovertebral dynamics, and the functionality of the respiratory centers.

The main objective is to analyze the anatomical and pathophysiological aspects and clinical repercussions that link MC-I, defined by the herniation of the cerebellar tonsils through the foramen magnum, and OSA. Compression of the brainstem is a key focus, since this region houses fundamental structures, such as the medulla and pons, where the respiratory centers responsible for generating and maintaining automatic breathing are located. The resulting dysfunction can cause respiratory instability, primarily predisposing to ACS, but it can also coexist with or aggravate OSA, due to changes in the neuromuscular control of the upper airways and deficits in respiratory coordination.

Through a critical literature review, the study investigates the anatomical origin of the symptoms and their clinical manifestations resulting from cerebellar herniation, also considering how such changes affect the biomechanics of breathing during sleep. Factors such as cervico-occipital pain, balance changes, autonomic dysfunctions, and cerebrospinal fluid flow disorders are correlated with functional impairments that directly impact the respiratory pattern, especially in sleep phases where ventilatory control depends mainly on automatic mechanisms.

In addition, the study analyzes the role of cardiorespiratory physical therapy in managing respiratory control dysfunctions and promoting improved sleep quality in patients with these interrelated conditions. Physical therapy intervention includes strategies aimed at

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optimizing ventilatory mechanics, re-educating breathing patterns, strengthening accessory muscles, and increasing functional capacity. The use of noninvasive ventilatory support, when indicated, and its contribution to stabilizing gas exchange and reducing apnea events, considering the anatomical and neurological specificities of MC-I, is also discussed.

Chiari malformation type I (CMI)

Chiari malformation type I (CM-I) is a congenital structural anomaly of the central nervous system that manifests itself in the craniovertebral junction. It is characterized by the herniation (or descent) of the cerebellar tonsils and lower structures of the cerebellum through the foramen magnum, an opening located at the base of the skull. The most widely accepted radiological criterion defines this descent as ≥ 5 mm in adults and ≥ 3 mm in children (Vagianou et al., 2022). Although predominantly congenital, the literature also describes acquired forms associated with intracranial pressure changes or trauma.

This anatomical change causes chronic compression of vital neural structures, mainly the upper brainstem and cervical spinal cord. The brainstem, which is composed of the midbrain, pons, and medulla, integrates motor, sensory, cognitive, and autonomic functions essential for survival (Kandel et al., 2021). It plays a critical role in controlling cardiorespiratory reflexes, fine motor coordination, balance, and as a passageway for all ascending and descending neural tracts.

The functional importance of the brainstem is even greater because it houses the respiratory and cardiovascular centers. These nuclei regulate automatic ventilatory rhythm, respiratory amplitude, blood pressure, and chemosensitive response to variations in CO_2 and blood pH (Guyton & Hall, 2021). Thus, any compression or deformity in this region, such as that which occurs in MC-I, can trigger neurological, autonomic, and respiratory dysfunctions of varying intensity.

In the respiratory sphere, the repercussions are particularly relevant. Compression of the bulbar nuclei can impair the transmission of the impulses necessary to initiate and maintain ventilation, promoting respiratory instability. This mechanism is directly related to ACS, characterized by the absence of respiratory effort due to central control failure. However, studies show that individuals with MC-I may also have OSA, since neuromuscular dysfunction of the upper airway and autonomic changes can contribute to pharyngeal collapse during sleep.

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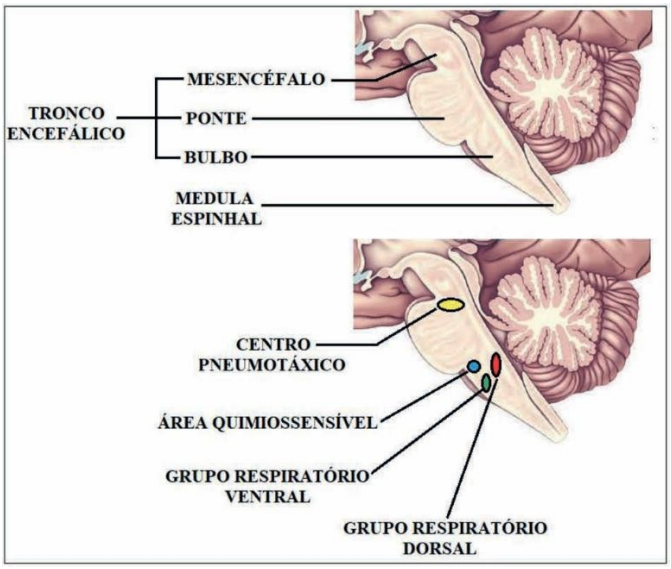
Figure 1. Illustrative diagram in two stages representing the anatomical and functional organization of the Respiratory Control Centers in the brainstem. The upper part identifies the Midbrain, Pons, Medulla, and continuity with the Spinal Cord. The lower part maps the vital respiratory nuclei: Dorsal Respiratory Group (DRG) and Ventral Respiratory Group (VRG), the Pneumotoxic Center, and the Central Chemosensitive Area.



Source: adapted from BEAR, 2017

SYMPTOMS OF CHIARI MALFORMATION TYPE I

The symptoms associated with Chiari Malformation Type I (CMI) result predominantly from compression of the neural structures located at the craniovertebral junction. Caudal herniation of the cerebellar tonsils through the foramen magnum compromises normal cerebrospinal fluid (CSF) circulation and can affect vital neurological areas. The variety and intensity of clinical manifestations are directly proportional to the degree of herniation and the level of compression imposed on adjacent structures, reflecting the functional complexity of this region.

Occipital headache is the most frequent symptom and is usually exacerbated by maneuvers that increase intracranial pressure, such as coughing, laughing, or squatting. Other common symptoms include neck pain, dizziness, imbalance, tinnitus, dysphagia, visual disturbances, and paresthesias in the upper limbs. In more severe cases, signs of autonomic dysfunction may occur, such as bradycardia, syncope, and respiratory disorders, including ACS, especially when the bulbar respiratory centers are compromised.

Motor manifestations, such as weakness or impaired motor coordination, may also arise due to interference with descending tracts and cerebellar function. In some individuals, CMT may be associated with syringomyelia, which aggravates the clinical picture by causing dissociated sensory loss, neuropathic pain, and additional motor impairment.

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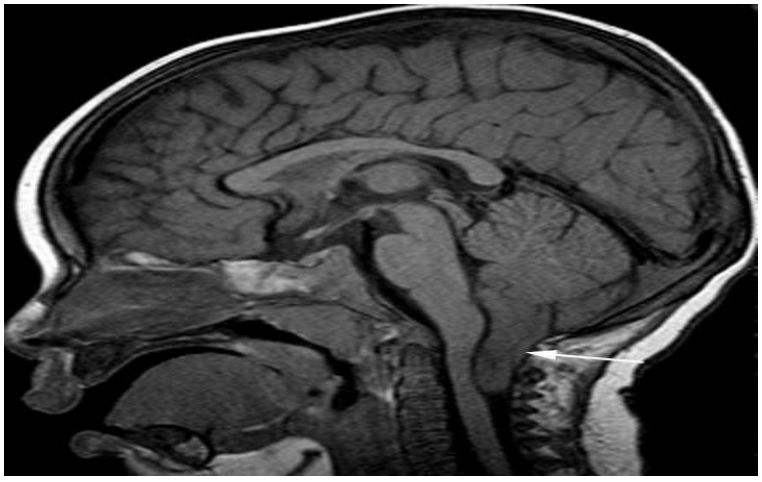
Although the focus of this study is limited to MCtI, it is crucial to distinguish between the different types of Chiari malformation. Each type has unique etiology, severity, and clinical manifestations, factors that significantly influence the diagnostic process and subsequent therapeutic management. Below is a comparative table containing the main types, their classifications, and respective symptoms, allowing for a broader understanding of the clinical variations involved.

1 Table: Shows the most relevant types, characteristics, and symptoms

TYPE	CHARACTERISTIC	SYMPTOMS
Type I	Described of cerebellar tonsils ≥5 mm below the foramen magnum. They are usually asymptomatic in childhood.	In adulthood, symptoms result from neural compression and altered CSF flow. They include: Headache in the back of the head and neck (worsened by exertion), pain and weakness in the arms and legs, dizziness/vertigo, imbalance, and sleep-disordered breathing (apnea).
Type II	Herniation of the cerebellum with part of the brain stem brain stem. Often associated with myelomeningocele (neural tube defect) and spina bifida, usually associated	Symptoms appear early in childhood and are linked to myelomeningocele and associated hydrocephalus. They include: partial or total paralysis below the spinal cord defect, swallowing problems and choking, laryngeal stridor (breathing noise), central apnea, and hydrocephalus.
Type III	Herniation of the cerebellum and brainstem outside the skull (encephalocele-hernia). High risk of mortality.	Due to extensive herniation and severe malformation of the central nervous system, symptoms are severe and lethal in most cases. These include: severe neurological deficits, paralysis, seizures, and high risk of death shortly after birth.
Type IV	Hypoplasia or absence of the cerebellum. Life-threatening in most cases.	Symptoms reflect extreme neurological impairment due to the absence of a structure vital for motor control and vital centers. The condition is generally incompatible with life (intrauterine death or death shortly after birth).

Image 2-Sagittal view on magnetic resonance imaging (MRI) of the brain, revealing Chiari Malformation Type 1 (CM1). Herniation of the cerebellar tonsils is observed, which are low and elongated (indicated by the arrow), clearly displaced below the level of the foramen magnum. This finding is characteristic of CMT1 and demonstrates compression at the craniovertebral junction.



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Source: UPTODATE via ESTRATÉGIA MED: Summary on Chiari Malformation Type 1: Definition, Clinical Manifestations, and More, n.d.).

RELATIONSHIP BETWEEN RESPIRATORY AND AND SLEEP (DRS) AND TYPE I CHIARIA MALFORMATION (MCt1)

The relationship between Sleep-Disordered Breathing (SDB) and Chiari Malformation Type I (CM-I) is widely recognized in the literature, mainly due to the direct interference of tonsillar herniation on structures crucial for automatic respiratory control. CM-I is characterized by the caudal displacement of the cerebellar tonsils through the foramen magnum, reducing the space available in the craniocervical junction and compressing essential regions of the brainstem, such as the medulla and pons. These structures house the respiratory centers responsible for generating spontaneous breathing, so their compression can compromise the conduction of impulses that initiate and maintain breathing, favoring episodes of ACS.

Although ACS is classically described as the predominant respiratory disorder in MC-I, recent studies show that AOSt can also occur with significant frequency in these patients. This is due to possible impairment of the lower cranial nerves — especially the glossopharyngeal (IX), vagus (X), and hypoglossal (XII) nerves — which actively participate in maintaining upper airway tone. Dysfunction of these nerves reduces pharyngeal stability, predisposing to mechanical collapse of the airway during sleep and contributing to obstructive or mixed events.

In addition to its direct effects on the respiratory centers and cranial nerves, CM-I can also interfere with cerebrospinal fluid (CSF) flow, affecting intracranial dynamics and autonomic modulation of breathing. Changes in CSF circulation are associated with ventilatory instability, variability in sensitivity to carbon dioxide levels, and fluctuations in respiratory rate.

Thus, the relationship between MC-I and RDS is multifactorial, resulting from a combination of structural compression, neurological dysfunction, and autonomic impairment. This complexity reinforces the need for detailed polysomnography to accurately identify the types of respiratory events present—central, obstructive, or mixed—and guide appropriate therapeutic strategies. (AOS, ACS, mechanisms, cranial nerves, bulbar compression.)

Central and obstructive mechanisms in Chiari malformation Type I

Chiari Malformation Type I (CMI) presents a complex set of anatomical and pathophysiological alterations capable of triggering different forms of Sleep-Related Breathing Disorders

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. Traditionally, its most associated manifestation is OSA, originating from direct dysfunction of the respiratory centers located in the brainstem. However, recent studies show that patients with cTMI can also develop OSA, resulting not only from pharyngeal structural factors, but also from neurological mechanisms related to compression of the lower cranial nerves. Thus, cTMI is characterized by a mixed respiratory pattern, resulting from the interaction between central and obstructive components.

The central component originates from compression of the bulbar structures. Herniation of the cerebellar tonsils reduces the space available at the craniocervical junction, compressing the medulla and pons in regions that house vital nuclei for respiratory control, such as the Dorsal Respiratory Group (DRG) and the Ventral Respiratory Group (VRG). These nuclei are responsible for generating the automatic respiratory rhythm and integrating impulses from the central chemoreceptors. When subjected to compression or stretching, there is impairment in neural transmission, leading to intermittent failures in the respiratory drive. This phenomenon manifests as respiratory pauses not preceded by ventilatory effort, a typical feature of ACS. In addition, changes in cerebrospinal fluid (CSF) flow in this region alter sensitivity to CO_2 levels, contributing to unstable breathing patterns and hypoventilation.

The obstructive component arises mainly due to neuropathy of the lower cranial nerves. The craniocervical junction is the point of origin of nerves IX (glossopharyngeal), X (vagus), and XII (hypoglossal), which play central roles in maintaining upper airway patency. The glossopharyngeal nerve participates in the sensory and motor control of the pharynx and is essential for protective reflexes. The vagus nerve is responsible for coordinating the vocal folds and laryngeal muscles, ensuring adequate opening of the glottis during breathing. The hypoglossal nerve controls the movements of the tongue, whose position determines pharyngeal stability. Compression of these nerves reduces the muscle tone of the upper airway, increasing susceptibility to pharyngeal collapse during sleep. In particular, during REM sleep, a phase marked by a physiological decrease in muscle tone, this vulnerability is amplified, favoring obstructive events.

When combined, these central and obstructive mechanisms result in mixed apneas, often observed in patients with MCtI. A respiratory event may begin as a central apnea due to failure of the ventilatory drive and progress to secondary obstruction if the pharyngeal muscles are unable to keep the airway open after the return of the respiratory impulse. This cycle perpetuates recurrent episodes of desaturation, micro-awakenings, and sleep fragmentation.

Therefore, understanding the mixed mechanism involving ACS and OSA in MCtI is essential to guide an accurate diagnosis and individualized treatment plan. Polysomnography is indispensable for identifying the predominant respiratory phenotype, while surgical decompression

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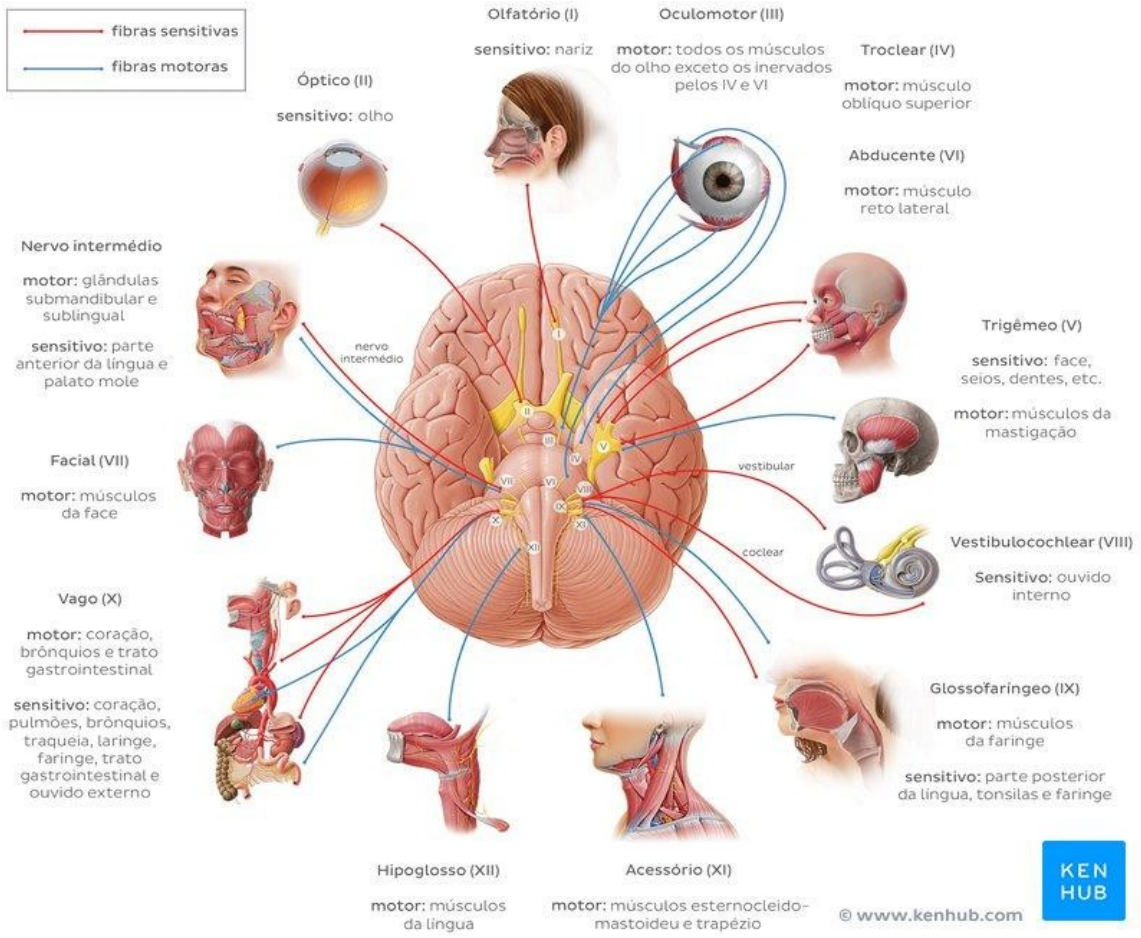
can, in many cases, restore neural flow and reduce both central and obstructive events.

Image 3: Schematic illustration of the base of the skull and brainstem, highlighting the origin of the lower cranial nerves. The image shows the path of the nerves:



Source: https://www.kenhub.com/pt/library/anatomia/os-12-nervos-cranianos

The dysfunction of these nerves due to compression exerted by T1MC prevents the muscles of the pharynx, larynx, and tongue from receiving the appropriate neural command to maintain their tone during sleep. The result is physical collapse and upper airway obstruction, characterizing OSA.

The clinical manifestation related to OSA in patients with MCt1 may be associated with other symptoms of cranial nerve dysfunction, such as dysphagia (affecting IX and X CN) and dysphonia (affecting X CN). It is for this reason that sleep-disordered breathing in patients with Chiari type I is often mixed, involving both failure of the central respiratory system signal and airway obstruction due to muscle weakness. Posterior fossa decompression (PFD) surgery, by relieving compression, can lead to improvement or resolution of apneas (OSA and CSA).

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PATHOPHYSIOLOGICAL MECHANISMS

Chiari malformation type I (CM-I) causes a series of structural and functional changes that directly affect breathing control and upper airway integrity during sleep. The main anatomical feature—caudal herniation of the cerebellar tonsils through the foramen magnum—results in mechanical compression of the brainstem, especially the medulla and pons, regions that house critical centers for the generation and modulation of respiratory rhythm. This compression can compromise the functioning of the pre-Bötzinger complex and central chemoreceptors, reducing the ventilatory response to CO_2 and favoring episodes of Central Sleep Apnea (CSA). As a consequence, respiratory instability, transient hypoventilation, and ventilatory pauses without inspiratory effort are observed.

In addition to central mechanisms, MC-I may also predispose individuals to the development of Obstructive Sleep Apnea (OSA). Compression of motor nuclei, such as the ambiguous nucleus and hypoglossal nucleus, interferes with neural control of the pharyngeal muscles responsible for upper airway patency. Pharyngeal hypotonia, intensified during REM sleep, increases the propensity for mechanical collapse of the airway. Autonomic disorders resulting from spinal cord and bulbar compression—including sympathetic and parasympathetic dysfunction—intensify ventilatory instability, favoring mixed respiratory events.

Cerebrospinal fluid dynamics also play an important role. Obstruction of cerebrospinal fluid flow in the foramen magnum can lead to increased intracranial pressure during certain phases of sleep, further altering the sensitivity of the respiratory centers. Episodes of intracranial hypertension can trigger fluctuations in ventilatory drive, contributing to complex respiratory events.

Additionally, cervical musculoskeletal changes—such as chronic pain, stiffness, and antalgic postural adaptations—modify the ventilatory pattern, which can reduce chest expansion and impair ventilation in the supine position. Thus, the pathophysiology of sleep-disordered breathing in MC-I is multifactorial, combining neuroanatomical compression, autonomic dysfunction, weakness of the upper airway muscles, and cerebrospinal fluid changes, which explains the frequent coexistence of ACS and OSA in these patients.

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Table 1. Pathophysiological mechanisms involved in MC-I and DRS

Type of Apnea	Primary Mechanism	Relationship with ICM
Central (ACS)	Respiratory drive failure, instability of the bulbar centers	Compression of the trunk stem and automatic
Obstructive (OSA)	Collapse of the upper airway during sleep	Pharyngeal neuromuscular dysfunction, changes in tone and intracranial pressure
Mixed	Combination of central and obstructive events	Consequence of the overlap of the above mechanisms

COMPRESSION OF THE BULBAR RESPIRATORY CENTERS

Compression of the bulbar respiratory centers is one of the main pathophysiological mechanisms responsible for the ventilatory instability observed in patients with Chiari Malformation Type I (CM-I). Herniation of the cerebellar tonsils through the foramen magnum significantly reduces the space available at the craniocervical junction, increasing mechanical pressure on essential structures of the brainstem. Among the most vulnerable areas are the Dorsal Respiratory Group (DRG), located in the region of the solitary tract nucleus, and the Ventral Respiratory Group (VRG), which integrates a neural network responsible for modulating and generating the automatic respiratory rhythm.

The DRG acts predominantly in the regulation of inspiration, through the integration of chemical and mechanical afferents from peripheral and central chemoreceptors. The VRG, on the other hand, is directly associated with the control of active expiration and the adjustment of ventilation in situations of increased demand. Compression of these regions compromises neural integration and the processing of respiratory stimuli, reducing sensitivity to serum CO_2 levels and making it difficult to maintain a stable respiratory rhythm during sleep.

As a result, temporary failure of the respiratory drive occurs, culminating in episodes of Central Sleep Apnea (CSA). In this condition, there is no ventilatory effort or attempt at chest expansion, since the primary neural stimulus is interrupted. The loss of automatic control triggers episodic hypoxemia, sleep fragmentation, and compensatory sympathetic hyperactivity. In the long term, this instability can contribute to secondary cardiovascular dysfunction, significant daytime fatigue, and a worsening quality of life.

Thus, bulbar compression is recognized as a central mechanism in the relationship between MC-I and central respiratory disorders.

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ALTERATION OF CEREBROSPINAL FLUID (CSF) FLOW

Altered cerebrospinal fluid (CSF) flow is one of the main pathophysiological mechanisms involved in Chiari Malformation Type I (CMI). Caudal herniation of the cerebellar tonsils through the foramen magnum reduces the space available at the craniocervical junction and can partially or totally obstruct the normal circulation of CSF between the cranial cavity and the cervical spinal canal. This mechanical obstruction directly interferes with craniocervical dynamics, creating an abnormal pressure gradient that compromises CSF absorption and pulsatile flow.

The alteration of cerebrospinal fluid flow also explains the frequent association between MC-I and syringomyelia, a condition characterized by the formation of an intramedullary cyst filled with CSF (syringes). When the passage of CSF through the foramen magnum is blocked or narrowed, intracranial pressure increases and the fluid tends to be diverted into the spinal cord, favoring the development of syringomyelia. This complication can intensify neurological deficits, including neuropathic pain, weakness, and sensory changes.

From a respiratory standpoint, blockage of CSF flow causes autonomic instability and interferes with the activity of the bulbar respiratory centers. The pressure change affects the signaling of central chemoreceptors responsible for detecting carbon dioxide (CO_2) levels and blood pH, reducing their sensitivity and impairing the adequate ventilatory response. As a result, the patient may experience irregular breathing patterns, episodes of hypoventilation, and a greater predisposition to ACS events.

In addition, pressure accumulation can amplify compression on the brainstem, aggravating neurological dysfunction and contributing to longer periods of respiratory instability, especially during sleep. Thus, altered CSF flow represents a critical component in the pathophysiology of MC-I, influencing both neurological and respiratory manifestations.

According to the American Association of Neurological Surgeons (AANS, 2020), about 1 in 1,000 people may have some degree of cerebellar tonsillar descent, but not all of them have symptoms.

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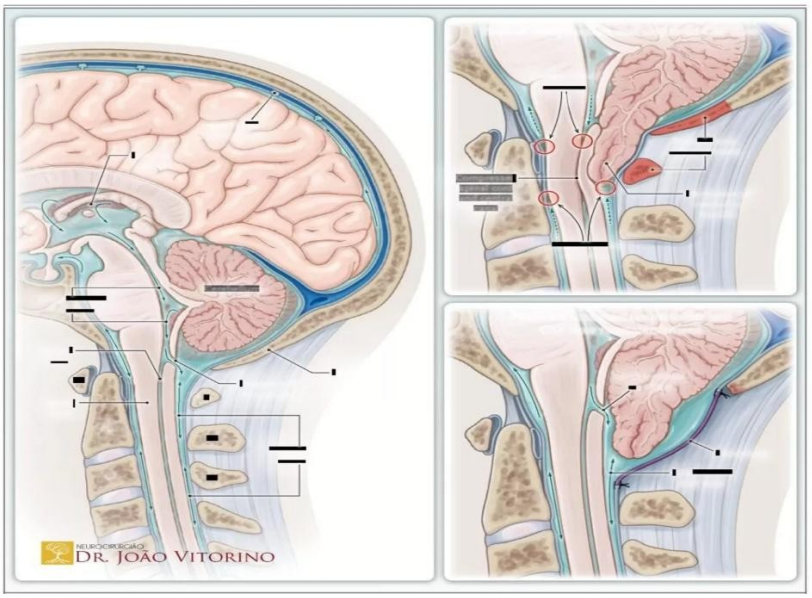
Figure 3 – *Schematic representation of the alteration in CSF flow caused by tonsillar herniation in Chiari Malformation Type I.*



Source: DR. JOÃO VITORINO (Neurosurgery).https://drjoaovitorino.com.br/malformacao-de-chiari-tipo-1-o-que-e-sintomas-e-quando-operar/

LOWER CRANIAL NERVE DYSFUNCTION

Lower cranial nerve dysfunction is one of the most relevant pathophysiological mechanisms in the relationship between Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB). Compression at the craniocervical junction can compromise the functioning of cranial nerves IX (glossopharyngeal), X (vagus), and XII (hypoglossal), all of which are directly involved in the motor and sensory coordination of the upper airway structures. These nerves control the pharynx, larynx, and tongue, playing a crucial role in maintaining pharyngeal tone and stability during sleep. When subjected to mechanical compression, their neural conduction is impaired, causing muscle weakness, loss of protective reflexes, and difficulty in supporting the airway walls.

The glossopharyngeal nerve (IX) participates in pharyngeal sensitivity and the swallowing reflex, while the vagus nerve (X) is responsible for the motor innervation of the larynx and the coordination of the vocal folds. The hypoglossal nerve (XII) controls the muscles of the tongue, which are central to the opening and positioning of the upper airway. The combined dysfunction of these nerves causes significant hypotonia, predisposing to pharyngeal collapse, especially during the REM phase of sleep, when physiological muscle tone is naturally reduced. This scenario favors the occurrence of OSA, often coexisting with central events resulting from bulbar compression.

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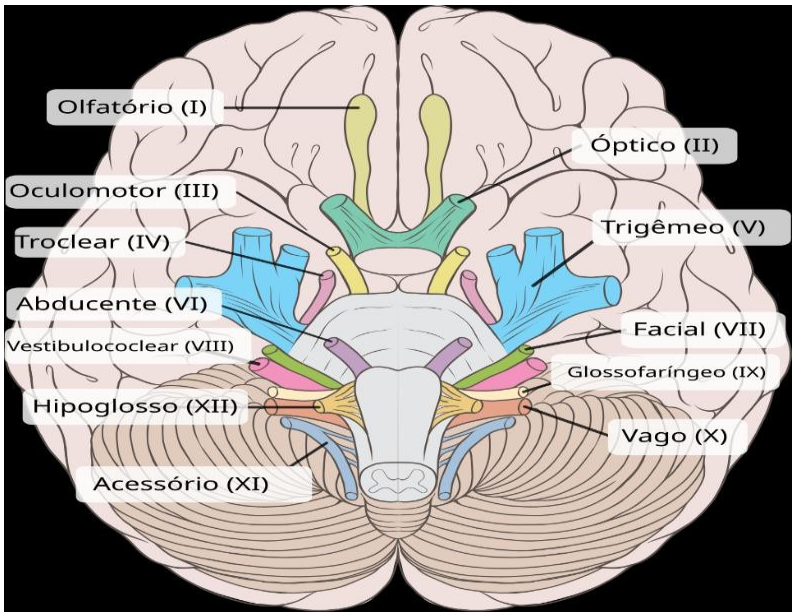
In addition, loss of sensitivity and reduced protective reflexes increase the risk of silent obstruction, hindering the body's perception of respiratory effort and aggravating sleep fragmentation. Thus, dysfunction of the lower cranial nerves represents a fundamental link in the pathophysiology of SUD associated with MC-I, directly influencing the transition between central, obstructive, and mixed events.

Figure 4 *– Origin and distribution of cranial nerves IX, X, and XII, highlighting their relationship with upper airway stability.*



Brainhumannormal inferior viewwithlabelsen.svg: Patrick J. Lynch, medical illustrator&derivativework : Beao *derivative work: Angelito7 (talk) - Brain_human_normal_inferior_view_with_labels_en.svg

DIAGNOSIS OF CHIARI MALFORMATION TYPE I (CMI)

The diagnosis of CTI is primarily based on clinical evaluation of symptoms and imaging tests.

Clinical Symptoms: Symptoms vary, but headache in the back of the head that worsens with exertion (coughing, sneezing) is the most common complaint in type I CMT, which usually manifests in adolescence or adulthood. Other signs may include neck pain, balance and coordination problems, dizziness, tinnitus, muscle weakness in the limbs, and, notably, sleep apnea or snoring.

Imaging (Gold Standard): Magnetic resonance imaging (MRI) of the skull and cervical spine is the definitive test. It allows visualization of the displacement of the cerebellar tonsils below the level of the foramen magnum (usually $\geq$ 5 mm), in addition to identifying associated complications, such as syringomyelia (cyst formation in the spinal cord).

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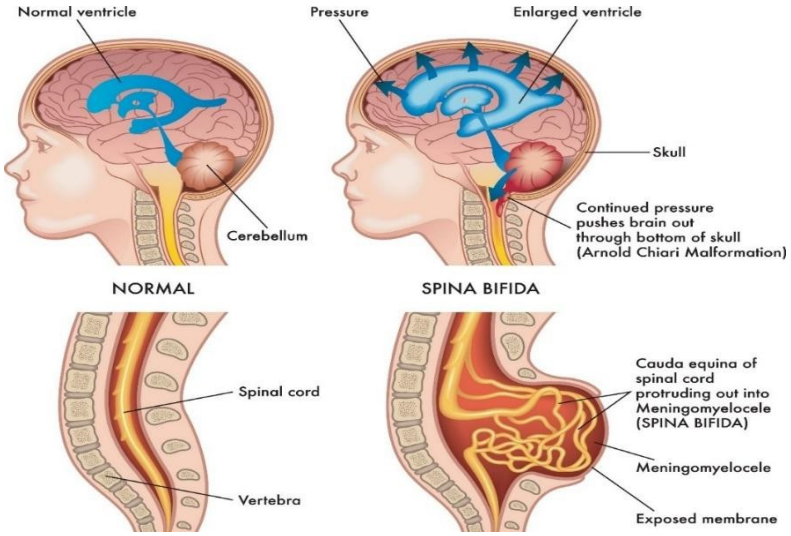

Source: Medical illustration

DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA (OSA)

OSA, characterized by recurrent episodes of partial or total obstruction of the upper airways during sleep, manifests as loud snoring, witnessed breathing pauses, and excessive daytime sleepiness.

Clinical Evaluation: This is performed through a detailed medical history, including the partner's report on snoring and breathing interruptions, and the application of questionnaires, such as the Epworth Sleepiness Scale. The physical exam may reveal anatomical abnormalities (such as a short jaw, enlarged tonsils or adenoids, increased neck circumference) that contribute to the obstruction.

Polysomnography (PSG) (Gold Standard): This is the essential test to confirm the diagnosis of OSA and quantify its severity. PSG monitors sleep and records the electroencephalogram (brain activity), electromyogram, electrooculogram, airflow, respiratory effort, oxygen saturation (pulse oximetry), heart rate, and snoring. The diagnosis of OSA is established by the Apnea-Hypopnea Index (AHI), which is the number of apnea (total cessation) and hypopnea (partial reduction) events per hour of sleep.

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Image 3- Representation of a patient being monitored during a polysomnography (PSG) exam in a sleep laboratory. The image illustrates the application of multiple sensors (electrodes and transducers) necessary for the simultaneous recording of physiological parameters during sleep, including: electrodes for monitoring brain activity (Electroencephalogram - EEG), electrodes for eye movement (Electro-oculogram - EOG), and airflow sensors (nasal and oral), as well as electrocardiogram (ECG) electrodes and respiratory bands (not specified in the source, but visible) for the detection of apnea/hypopnea events and the assessment of sleep architecture.

Source: https://www.google.com/search?q=telemedicinamorsch.com.br/blog/exame-de-polissonografia

THE RELATIONSHIP OF THE COMBINED APPROACH

A combined approach is essential in the management of Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB), since both conditions share interconnected pathophysiological mechanisms. The tonsillar herniation characteristic of CM-I causes compression of the brainstem and bulbar structures responsible for controlling breathing, directly interfering with the functioning of the centers that regulate respiratory rhythm. This alteration is recognized as one of the main causes of Central Sleep Apnea (CSA). However, it is not uncommon for patients to simultaneously present typical characteristics of Obstructive Sleep Apnea

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, or even a predominance of the obstructive component. This results from dysfunction of the lower cranial nerves, especially IX and X, which are responsible for the muscle tone of the pharynx and upper airway muscles. Thus, while the central component is linked to the interruption of respiratory neural commands, the obstructive component results from the mechanical collapse of the upper airway during sleep.

These pathophysiological overlaps explain why an isolated approach is rarely sufficient. Therapeutic success depends on understanding how structural damage resulting from MC-I interacts with anatomical and functional factors that favor pharyngeal collapse. Thus, the combination of neurosurgical, respiratory, and rehabilitative interventions becomes not only recommended but essential to restore ventilatory stability and improve the patient's quality of life.

The Need for a Multidisciplinary Approach

The central point of the combined approach is a **complete diagnostic evaluation**, followed by treatment tailored to the patient's specific needs. In this context, polysomnography (PSG) plays a key role. In addition to confirming the presence of OSA and quantifying its severity using the Apnea-Hypopnea Index (AHI), PSG allows for accurate differentiation between central, obstructive, and mixed events. This distinction is crucial, as it determines whether the initial focus will be neurosurgical intervention, ventilatory support, or functional rehabilitation.

The differential diagnosis also prevents inappropriate treatments, such as prescribing CPAP in a patient whose apnea is predominantly central, or performing decompressive surgery in cases where OSA is clearly primary. Thus, PSG guides therapeutic individualization and allows different professionals to work together synergistically.

In addition to PSG, imaging tests, such as magnetic resonance imaging (MRI) of the skull and cervical region, complement the diagnostic process by revealing the degree of tonsillar herniation, changes in cerebrospinal fluid flow, the presence of syringomyelia, or marked bulbar compression. The correlation between clinical, respiratory, and imaging findings helps to define the most appropriate therapeutic path.

Integration between Neurosurgery, Sleep Medicine, and Physical Therapy

When ACS or significant bulbar dysfunction predominates, **posterior fossa decompression (PFD)** is considered the primary treatment. The surgery aims to restore normal cerebrospinal fluid dynamics and relieve pressure on the brainstem, often resulting in a reduction

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or complete resolution of central events. However, many patients maintain OSA after PFD, demonstrating that surgery, although necessary, is not sufficient to correct all respiratory components. Therefore, after the procedure, PSG evaluation is repeated to guide complementary management.

In cases of persistent or predominant OSA, therapies such as **CPAP**, **BPAP**, intraoral devices, or servo-adaptive ventilation come into focus. These interventions maintain the upper airway patency during sleep, preventing recurrent collapses.

Physical therapy plays an essential role in the integrated approach. Respiratory physical therapy optimizes ventilatory mechanics, improves breathing patterns, and contributes to strengthening respiratory muscles. Orofacial myofunctional physical therapy acts directly on the tone of the upper airway, strengthening the suprahyoid muscles, tongue, masseter, and soft palate—a particularly effective intervention in cases of OSA. The physical therapist also works on postural aspects, which are fundamental in patients with MC-I who have cervical stiffness or suboccipital pain, which impacts breathing.

INTEGRATED TREATMENT PLAN WITH EMPHASIS ON PHYSIOTHERAPY

The integrated treatment plan for patients with Chiari Malformation Type I (CM-I) associated with Sleep-Disordered Breathing (SDB) must consider the pathophysiological complexity of this condition, in which central, obstructive, and neuromuscular mechanisms coexist and directly influence ventilation. Thus, the therapeutic strategy must be individualized, ranging from the correction of structural factors to the functional management of respiratory disorders. The definition of the plan depends on the clinical presentation, neurological severity, imaging findings, and polysomnographic profile, allowing for a more accurate and effective approach.

Treatment of MC-I becomes a priority when there are clinical or polysomnographic signs of Central Sleep Apnea (CSA), evidence of significant bulbar compression, or the presence of neurological manifestations such as typical occipital headache, dysphagia, dizziness, autonomic changes, muscle weakness, and balance disorders. Posterior fossa decompression (PFD) is the primary intervention for these cases. Its goal is to increase the space available for the structures of the craniocervical junction, restoring adequate cerebrospinal fluid flow and reducing pressure on the brainstem, especially in the bulbar region, where vital respiratory control centers are located. Clinical studies indicate that PFD results in significant improvement in ACS and associated neurological symptoms, with a positive impact on overall functionality. However, the effectiveness of surgery in Obstructive Sleep Apnea (OSA) is variable, justifying the need for

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Continuous polysomnographic monitoring in the postoperative period to assess possible residual changes.

When OSA is predominant, or when it persists after surgical correction, the approach should include therapies aimed at upper airway patency. Continuous Positive Airway Pressure (CPAP) ventilation remains the first choice in the treatment of residual OSA due to its effectiveness in preventing pharyngeal collapse during sleep. In cases of CPAP intolerance or in patients with significant oropharyngeal muscle weakness, alternative modalities such as BPAP, servo-adaptive ventilation, backup frequency devices, or intraoral mandibular advancement devices should be considered. These strategies not only favor maintaining an open airway but also offer adequate ventilatory support in cases of hypoventilation.

The Role of Physical Therapy in Integrated Treatment

Physical therapy plays an essential role in multidisciplinary treatment plans, acting both in the preoperative phase and after surgery, in addition to being fundamental in the management of OSA and cardiorespiratory rehabilitation. In the context of CM-I, the physical therapist contributes to the optimization of ventilatory mechanics and improvement of respiratory function through interventions such as chest expansion exercises, techniques to increase chest compliance, costovertebral mobilization, diaphragmatic training, and strengthening of the respiratory muscles. Such interventions enhance alveolar ventilation, reduce dysfunctional breathing patterns, and minimize the occurrence of respiratory muscle fatigue.

In addition, the focus of physical therapy on postural reeducation plays a decisive role for patients with CM-I, since improper cervical posture can increase suboccipital muscle tension and compromise respiratory mechanics. Cervical stabilization exercises, posterior muscle stretching, and myofascial release techniques contribute to the reduction of occipitocervical pain and the proper biomechanical alignment of the cervical spine.

In patients with OSA, orofacial myofunctional therapy is crucial for increasing the tone of the suprahyoid muscles, tongue, soft palate, and lateral walls of the pharynx. Strengthening these structures helps prevent pharyngeal collapse during sleep, improves breathing patterns, reduces snoring, and increases respiratory efficiency. Combined with sleep hygiene, respiratory control techniques, and specific breathing exercises, myofunctional therapy has been shown to be effective in reducing the severity of OSA and in adapting to the use of CPAP.

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The integration between neurosurgeons, neurologists, pulmonologists, otolaryngologists, physical therapists, and sleep medicine specialists is the central axis of MC-I treatment with DRS. The multidisciplinary approach allows for the identification of the dominant etiology—neural compression, musculoskeletal alteration, or pharyngeal instability—and the targeting of combined therapies that increase treatment efficacy, prevent complications, and promote a better quality of life. The integrated approach ensures that each aspect of the condition is treated synergistically, guaranteeing greater safety, functionality, and overall recovery for the patient.

PHYSIOPATHOLOGICAL THEORY OF THE ASSOCIATION BETWEEN MCt1 AND DRS

The relationship between Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB) has been a topic of growing clinical and scientific interest due to the high prevalence of central and obstructive apneas in individuals with tonsillar herniation. Several pathophysiological theories have been proposed over the last few years, and although they differ from one another, they are often complementary, reflecting the structural and functional complexity of the craniocervical junction.

The first theory, and probably the most widely recognized, is based on direct compression of the bulbar structures involved in the automatic generation of the respiratory rhythm. The reduction of space in the foramen magnum can affect nuclei such as the Dorsal and Ventral Respiratory Groups, responsible for capturing chemical signals and modulating respiratory movements. Such compression compromises the neuronal drive, reduces chemoreflex sensitivity to CO_2, and favors episodes of Central Sleep Apnea (CSA), characterized by the absence of ventilatory effort. From this perspective, the greater the herniation, the greater the risk of central respiratory dysfunction.

Another hypothesis highlights the participation of neuromuscular dysfunction of the upper airway. Compression of pharyngeal motor nuclei, such as the ambiguous nucleus and the hypoglossal nucleus, can cause an imbalance in the muscle tone of the pharyngeal dilators, especially during REM sleep. This mechanism leads to critical narrowing or intermittent collapse of the upper airway, favoring Obstructive Sleep Apnea (OSA). The coexistence of ACS and OSA in patients with MC-I reinforces the multisystemic nature of the condition.

A third theory considers changes in cerebrospinal fluid dynamics to be a key factor. Partial obstruction of cerebrospinal fluid (CSF) circulation in the foramen magnum can cause episodic intracranial hypertension and transient changes in bulbar function. Studies suggest that this pressure fluctuation may influence the respiratory pattern, inducing ventilatory instability, periodic breathing, and mixed apneas.

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Finally, biomechanical and autonomic factors are also considered relevant contributors. Chronic cervical pain, limited chest expansion, and dysautonomia secondary to spinal cord compression can accentuate respiratory irregularities during sleep.

Thus, it is understood that the association between MC-I and RSD results from a combination of neuroanatomical, muscular, and cerebrospinal fluid mechanisms that act in an integrated manner. No single theory can fully explain the phenomenon, reinforcing the need for a multidimensional approach in the evaluation and clinical management of these patients.

EPIDEMIOLOGY

Chiari Malformation Type I (CM-I) is a rare anomaly of the craniocervical junction characterized by herniation of the cerebellar tonsils through the foramen magnum. Its global prevalence varies greatly depending on population access to magnetic resonance imaging (MRI). In the United States, prevalence is estimated to be between **0.5% and 1%**, while European countries report values between **0.3% and 0.8%**, reflecting methodological and demographic differences. The symptomatic incidence, that is, individuals who present neurological symptoms and seek medical care, remains universally low, ranging from **0.7 to 0.9 cases per 100,000 inhabitants/year**.

Epidemiology shows **a** strong **female predominance**, possibly associated with anatomical, hormonal, and connective tissue factors. Most diagnoses occur between **the ages of 15 and 45**, when symptoms such as occipital headache, paresthesia, and autonomic disorders become more evident. Despite this, a large proportion of patients remain asymptomatic for years, which contributes to late diagnosis.

In contrast, Obstructive Sleep Apnea (OSA) is a prevalent respiratory disorder characterized by recurrent collapses of the upper airways during sleep. OSA has a much higher prevalence: about **4% of the adult population**, reaching 10% in some groups. Its annual incidence is estimated at **30 cases per 100,000 inhabitants**, exceeding the symptomatic incidence of MC-I by almost 40 times.

As with MC-I, there is greater involvement in postmenopausal women, but in the general population, OSA is more common in men. Both conditions present significant risks: MC-I can progress to neurological deficits and central respiratory disorders, while OSA is associated with increased cardiovascular mortality, as described in the classic literature.

The epidemiological comparison between MC-I and OSA highlights the discrepancy between a **rare structural neurological disease** and a **highly prevalent respiratory disorder**, but both

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have a significant impact on quality of life and respiratory risk. The graphs below illustrate the differences in prevalence and incidence between the two conditions

Epidemiological Variable	Malformation of Type I (CMI)	Obstructive Sleep Apnea (OSA)
Prevalence	0.3% – 1.0%	~4% (can reach 10%)
Incidence (annual)	0.7–0.9 / 100,000 inhabitants	~30 / 100,000 inhabitants
Predominance by gender	Higher in women	Higher in men (except postmenopausal)
Typical age range	15–45	40–70 years
Type of condition	Structural neurological	Functional respiratory

MATERIAL

This study was based on an integrated narrative review of the literature, developed with the aim of identifying, analyzing, and synthesizing scientific information about the relationship between Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB), with an emphasis on Obstructive Sleep Apnea (OSA). Scientific materials published between 2019 and 2025 were selected, a period in which studies involving advanced neuroimaging, digital polysomnography, and standardized respiratory assessment protocols became more frequent and robust.

The material used comprised original articles, systematic reviews, clinical consensus statements, international guidelines, book chapters, and documents from renowned scientific associations, such as the American Academy of Sleep Medicine (AASM) and the American Association of Neurological Surgeons (AANS). Databases such as PubMed, SciELO, ScienceDirect, and Google Scholar were consulted using standardized descriptors (DeCS/MeSH), including: “Chiari Malformation Type I,” “Sleep Apnea, Obstructive,” “Sleep Apnea, Central,” “Sleep Disorders,” “Brainstem Compression,” “Cranial Nerve Dysfunction,” and “Posterior Fossa Decompression.”

Inclusion criteria covered studies:

published in English, Portuguese, or Spanish;

involving pediatric or adult patients with a confirmed diagnosis of CM-I by magnetic resonance imaging;

evaluating respiratory parameters by polysomnography;

describing pathophysiological mechanisms, neurological findings, prevalence, or therapeutic impact.

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Materials related to other malformations (Chiari II–IV), articles without access to the full text, studies with inadequate methodology, and case reports without pathophysiological relevance were excluded.

The material selection process was structured in three stages: initial screening by title, reading of abstracts, and finally, complete reading of texts that met the proposed criteria. To ensure scientific rigor, articles with a higher level of evidence, such as observational clinical trials, systematic reviews, and meta-analyses, were prioritized.

In addition, representative scientific images were used as part of the supplementary material, including anatomical diagrams of the craniocervical transition, inferior cranial nerve pathways, and cerebrospinal fluid flow changes. These visual elements allowed for a better understanding of the biomechanical relationships involved in ICM.

At the end of the screening and analysis, the set of selected materials provided a solid basis for understanding the multiple mechanisms of the association between ICM and DRS, enabling a critical and structured approach to the results presented in this study.

METHOD

This study was based on an integrated narrative review of the literature, developed with the objective of identifying, analyzing, and synthesizing scientific information about the relationship between Chiari Malformation Type I (CMI) and Sleep-Disordered Breathing (SDB), with an emphasis on Obstructive Sleep Apnea (OSA). Scientific materials published between 2019 and 2025 were selected, a period in which studies involving advanced neuroimaging, digital polysomnography, and standardized respiratory assessment protocols became more frequent and robust.

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evaluating respiratory parameters by polysomnography;

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At the end of the screening and analysis, the set of selected materials provided a solid basis for understanding the multiple mechanisms of the association between MC-I and DRS, enabling a critical and structured approach to the results presented in this study.

SUMMARY

The analysis of the selected studies showed that the prevalence of Sleep-Disordered Breathing (SDB) in individuals with Chiari Malformation Type I (CMI) varies between 35% and 70%, depending on the population studied, age, and polysomnographic criteria used. It was observed that both Central Sleep Apnea (CSA) and Obstructive Sleep Apnea (OSA) are frequent among these patients, evidencing the multifactorial nature of the condition.

The results indicated that CSA is more frequently associated with bulbar compressions, especially in children. In adults, however, there is an increasing prevalence of obstructive events, suggesting that lower cranial nerve dysfunction and loss of pharyngeal tone play a significant role. Polysomnography revealed that many patients have mixed respiratory events, characterized by a succession of central pauses followed by upper airway collapse.

Analysis of magnetic resonance images confirmed that the degree of tonsillar herniation alone does not determine the severity of RSD. Studies have shown that complementary changes, such as syringomyelia, narrowing of the subarachnoid space, and impaired cerebrospinal fluid flow, directly influence respiratory physiology.

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Regarding therapeutic impact, posterior fossa decompression resulted in substantial improvement in central events in most studies, with regression rates ranging from 60% to 90%. However, the response of OSA was heterogeneous: some patients showed partial improvement, while others continued to have high obstruction indices after the procedure. This indicates that OSA may result from persistent dysfunction of the cranial nerves responsible for controlling the airway muscles.

The use of CPAP has been shown to be effective for controlling residual OSA, especially in individuals with persistent hypotonia or structural irregularities. In more complex cases, ventilatory modalities with frequency backup were necessary, especially when central events coexisted.

In summary, the results suggest that MC-I is a relevant risk factor for DRS and that the respiratory phenotype is determined by the interaction between bulbar compression, cerebrospinal fluid changes, and pharyngeal neuromuscular dysfunction.

DISCUSSION

This review reveals that the association between Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB) is more complex than previously recognized, involving central and obstructive mechanisms that coexist and influence each other. The findings suggest that compression of the brainstem, particularly the medulla, remains the main determinant of Central Sleep Apnea (CSA). This compression interferes with the neural signaling responsible for the automatic maintenance of ventilation, impairing the response to CO_2 levels and the generation of respiratory drive.

On the other hand, the increasing presence of Obstructive Sleep Apnea (OSA) in patients with MC-I reinforces the importance of lower cranial nerve dysfunction. By compromising the motor control of the pharynx and tongue, these neuropathies reduce the tone of the upper airway, facilitating collapse during sleep, especially in phases of lower muscle tone, such as REM sleep. This interaction between central instability and mechanical obstruction explains the high prevalence of mixed respiratory events observed in studies.

Another important point is the variability of postoperative results. Posterior fossa decompression has been shown to be effective in reducing central events, but it does not show the same consistency in the treatment of OSA. This suggests that decompression relieves pressure on respiratory centers but does not fully reverse preexisting neuromuscular damage, especially in cases of chronic compression.

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In addition, the findings confirm that the severity of RSDs does not depend exclusively on the degree of tonsillar herniation, but rather on a set of anatomical and functional factors, including cerebrospinal fluid flow, changes in posterior fossa volume, and individual variations in chemoreflex sensitivity.

In this context, the need for a multidisciplinary approach involving neurosurgery, sleep medicine, neurology, and cardiorespiratory physical therapy is emphasized. Polysomnography should be performed in all patients with symptomatic CRS, both pre- and postoperatively, ensuring accurate diagnosis and continuous management.

In summary, the discussion shows that MC-I should be understood as a systemically integrative neurological condition that directly impacts respiratory physiology and requires personalized interventions.

FINAL CONSIDERATIONS

This review has allowed us to consolidate a comprehensive set of evidence on the relationship between Chiari Malformation Type I (CM-I) and Sleep-Disordered Breathing (SDB), especially Obstructive Sleep Apnea (OSA) and Central Sleep Apnea (CSA). The findings demonstrate that CM-I should be understood as a complex neurological condition, whose impact goes beyond structural changes in the craniocervical junction and affects fundamental physiological systems, such as ventilatory control, autonomic regulation, and upper airway tone. The coexistence of central and obstructive mechanisms reinforces the multifactorial nature of SUD in this group of patients, which requires detailed investigation and a multidimensional therapeutic approach.

Polysomnography (PSG) remains the fundamental diagnostic method for accurately differentiating central, obstructive, and mixed events, allowing for a more complete characterization of the respiratory phenotype and guiding individualized treatment. Posterior fossa decompression (PFD) has shown positive results, mainly in reducing central events, confirming the relevance of bulbar compression as a critical pathophysiological mechanism of ACS. However, the persistence of OSA after the procedure in a significant portion of patients indicates that neuromuscular changes may remain even after anatomical correction, reinforcing the need for complementary therapies, such as CPAP or advanced ventilatory modalities.

Another important aspect revealed is the independence between the degree of tonsillar herniation and the severity of RSD, suggesting that factors such as cerebrospinal fluid flow, lower cranial nerve dysfunction, and posterior fossa volume changes play a decisive role. This finding reinforces that MC-I should not be evaluated solely by tonsillar displacement, but by a set of clinical, neurological, and respiratory elements.

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The results discussed point to the urgent need for more standardized clinical protocols, including systematic screening for RSD in patients with MC-I, especially those with symptoms such as dysphagia, snoring, daytime fatigue, and cervico-occipital pain. Integration between neurosurgeons, neurologists, pulmonologists, physical therapists, and sleep medicine specialists is essential to ensure early diagnosis and appropriate management.

It is concluded that the association between MC-I and DRS represents a field of growing relevance that is still underexplored. Early detection, complete polysomnographic evaluation, and multidisciplinary treatment offer the best prospects for reducing complications, improving functionality, and promoting quality of life. Future research, with more robust methodologies and larger samples, is essential to clarify remaining gaps and optimize therapeutic strategies.

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