ABDUCTOR PARALYSIS- CASE PRESENTATION WITH HOMOEOPATHIC MEDICAMENT

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Introduction:

Bilateral vocal fold (vocal cord) immobility (BVFI) is a broad term that refers to all forms of reduced or absent movement of the vocal folds. Bilateral vocal fold (cord) paralysis (BVFP) refers to the neurological causes of bilateral vocal fold immobility (BVFI) and specifically refers to the reduced or absent function of the vagus nerve or its distal branch, the recurrent laryngeal nerve (RLN). Vocal fold immobility may also result from mechanical derangement of the laryngeal structures, such as the cricoarytenoid (CA) joint.

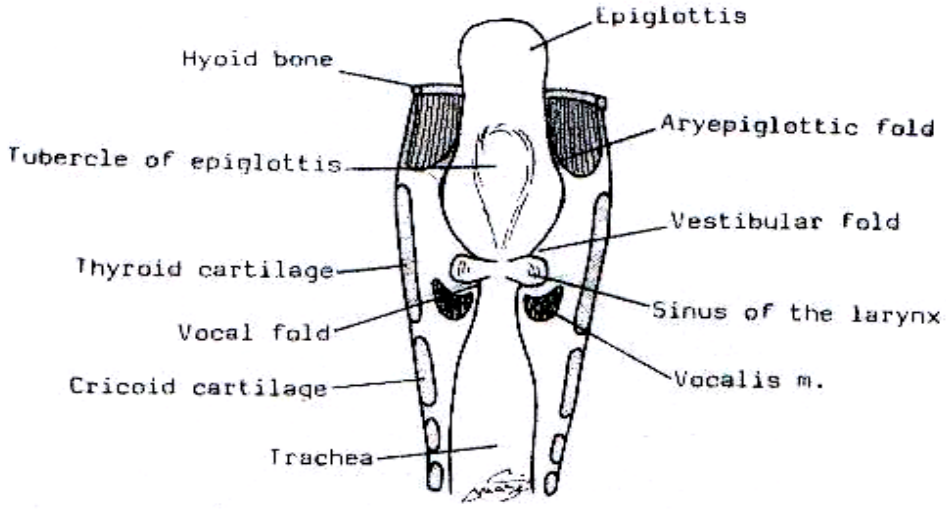


Fig.5. the larynx coronal section

Although a small number of conditions account for most cases of vocal cord immobility, this paper presents a comprehensive knowledge on clinical presentations, diagnostic workup, and treatment options. The goal of the paper is to provide the clinician with a basic understanding of the rare entity of bilateral vocal fold immobility (BVFI) with Homoeopathic approach.

Definition:

Vocal cord paralysis is caused by paralysis of intrinsic muscles of larynx. This is a symptom of an underlying disorder and not a disease by itself. The intrinsic muscles of the vocal cord are supplied by the vagus nerve. The term vagus means "wanderer" which is the apt term to describe this nerve because of its long anatomical course.

Unilateral vocal fold paralysis occurs due to dysfunction of recurrent laryngeal or vagus nerve causes a breathy voice. The breathiness of voice is caused by glottic chink, which allows air to escape when the patient attempts to speak. Normal voice production is dependent on proper glottal closure resulting from bilateral adduction of the vocal cords. This adduction of vocal folds combined with subglottic air pressure causes the vocal folds to vibrate causing phonation.

Incidence:

The incidence of vocal cord paralysis in the general population is not known. In infants and children, vocal cord paralysis accounts for 10% of all congenital anomalies of the larynx; second only to laryngomalacia. Left cord is paralyzed more frequent than right (2:1), due to its longer course.

Etiology:

Vocal cord paralysis may be congenital or acquired.

Congenital vocal cord palsy: Many infants with stridor may have congenital paralysis of vocal cords. This could occur with or without other associated abnormalities (i.e. neurological, laryngeal and cardiac defects). The most commonly associated anomaly in these patients is the presence of hydrocephalus. The mechanisms of vocal cord palsy in these children are still not clear. It could be due to stretching of the vagus nerve, due to complicated delivery etc.

Acquired causes of vocal cord palsy:

Table showing the probable acquired causes of vocal cord palsy along with their incidences:

Causes of vocal cord palsy	Percentage
Malignant disease	31%
Surgical trauma	29%
Idiopathic	24%
Non surgical trauma	7%
Inflammatory	4%
Neurological	1%
Miscellaneous	4%

Malignant disease*:* One third of all vocal cord paralysis is caused by malignancies like lung cancer, esophageal cancer and thyroid malignancies. Other rare causes could include temporal lobe malignancies, posterior fossa tumors, paragangliomas etc.

Surgical trauma is the second commonest cause of vocal cord paralysis. Thyroid surgeries are the commonest. Mediastinal surgeries, esophageal surgeries can also cause vocal cord palsy.

Nonsurgical trauma: Injuries to neck caused by automobile accidents and penetrating neck injuries can cause vocal cord palsy.

Inflammatory causes: By far the most common cause is tuberculosis. This could be due to apical scarring of the mediastinum or enlargement of hilar nodes. Other rare causes include jugular vein thrombophlebitis following csom, subacute thryoiditis, and meningitis both viral and bacterial.

Neurologic causes: Include brain stem ischemia, multiple sclerosis and head injuries.

Miscellaneous causes: include hemolytic anemia, thrombosis of subclavian vein, syphilis, collagen disorders, lead and arsenic poisoning.

Idiopathic causes: A major chunk of the recurrent laryngeal nerve paralysis fall under this group where in no demonstrable abnormality could be attributed to recurrent laryngeal nerve paralysis. Left vocal cord is commonly involved in these patients. Many of the idiopathic recurrent laryngeal nerve paralysis is caused by viral infections (sub clinical). Recovery is common in these patients.

Pathophysiology:

Although a comprehensive discussion of each of the causes is beyond the scope of this article, some principles should be emphasized. With the first episode of bilateral vocal fold paralysis (BVFP), patients may have dysphonia because the vocal cords are too far apart. Over time, however, the vocal cords can move to a medial position, and the patient may have a good voice and cough despite stridor and bilateral vocal fold paralysis (BVFP). As the vocal cords migrate toward the midline, the voice (and cough) improves, while the airway worsens. Clinicians should not mistake a good voice and cough as signs of a functioning larynx, especially in a patient with stridor. Aspiration and dysphagia may or may not be present in patients with vocal cord paralysis.

Pathogenesis of vocal cord paralysis:

Vocal cord paralysis is a sign of disease and is not a diagnosis. It may be due to a lesion anywhere from the cerebral cortex to the neuromuscular junction. Because of the large size of the nucleus ambiguous, small lesions in it may produce isolated laryngeal and pharyngeal motor losses. Lesions involving the nucleus ambiguous may cause bilateral paralysis more often than unilateral palsy.
Peripheral damage to the laryngeal innervations may be of three types:

1. Damage to the vagus trunk above the nodose ganglion, the origin of superior laryngeal nerve

2. Damage to the vagus nerve below the level or to the recurrent laryngeal nerve

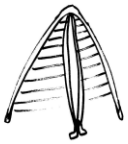
3. Damage to the superior laryngeal nerve alone.

***Clinical Feature*:**

Depends on position of paralyzed cord. So, we may get:

A. **Unilateral abductor paralysis (paralyzed cord in the median position):** Some dyspnoea under the stress of exercises.
B. **Unilateral adductor paralysis (paralyzed cord in the intermediate position):** voice is breathy (hoarseness), and there may be aspiration.

C. **Bilateral abductor paralysis (paralyzed cords in median position):** severe airway obstruction and stridor, but good voice.



D. **Bilateral adductor paralysis (paralyzed cords in intermediate position):** Aphonia and severe aspiration. This is fatal from the cause or from aspiration.
E. **Superior laryngeal nerve paralysis:** Loss of high-pitched voice and temporary aspiration.

Diagnosis:

The standard diagnostic workup and evaluation of a patient with vocal cord paralysis of unknown etiology is as follows: CXR, cervical spine series, barium swallow, thyroid scan, CT or MRI of head, neck, and possibly thorax, CBC, Thyroid function tests, ESR, Rheumatoid factor, Parathyroid hormone, calcium and glucose levels, PPD, VDRL, fungal titers, lyme titers, and possibly a lumbar puncture.

From the history and clinical picture.

Examination of the larynx: by mirror, rigid telescope or flexible nasopharyngolaryngoscopy.

Radiological evaluation: for the neck, chest and skull base, usually by CT.

Laboratory studies: for diabetes, TB, and syphilis.

***Complications*:**

1. Airway compromise
2. Wound infection
3. Hematoma
4. Extrusion of the implant
5. Laryngocutaneous fistula formation

Treatment option as advocated by modern medicine:

1) Unilateral abductor paralysis: Speech therapy.
2) Unilateral adductor paralysis: Intracord injection (as Teflon) or medilization thyroplasty.
3) Bilateral abductor paralysis: Arytenoidectomy (endoscopic or external), lateralization, or cordotomy (by cold instruments, radiofrequency or laser). Tracheostomy is usually needed as a life saving procedure.
4) Bilateral adductor paralysis: laryngeal diversion or laryngectomy.
5) Superior laryngeal nerve paralysis: Speech therapy.

Treatment option with Homoeopathic Therapy:

As Homoeopathy deals with the patient but not with the disease, we have to adhere to the dictum as advocated by the stalwarts, erudite scholars and veterans of Homoeopathy. As we know to study the drug and disease symptoms are the media for every scientific system. It is seen from all literature that Hahnemann advocated following fundamentals, which are accepted universally by all.

a. Nothing can be known of disease except symptoms.
b. It is patient who is ill and not his parts or organs.
c. Symptoms furnish the only unfailing guide for selection of remedy.
d. The remedy is hardly ever indicated by a single symptom however peculiar it may be.
e. Peculiar, characteristics, individualizing symptoms in the case and not common symptoms denote similimum.

Homoeopathy chooses the similimum by basis of the totality of symptoms, which has been applied in our case and it is presented below.

Case Presentation:

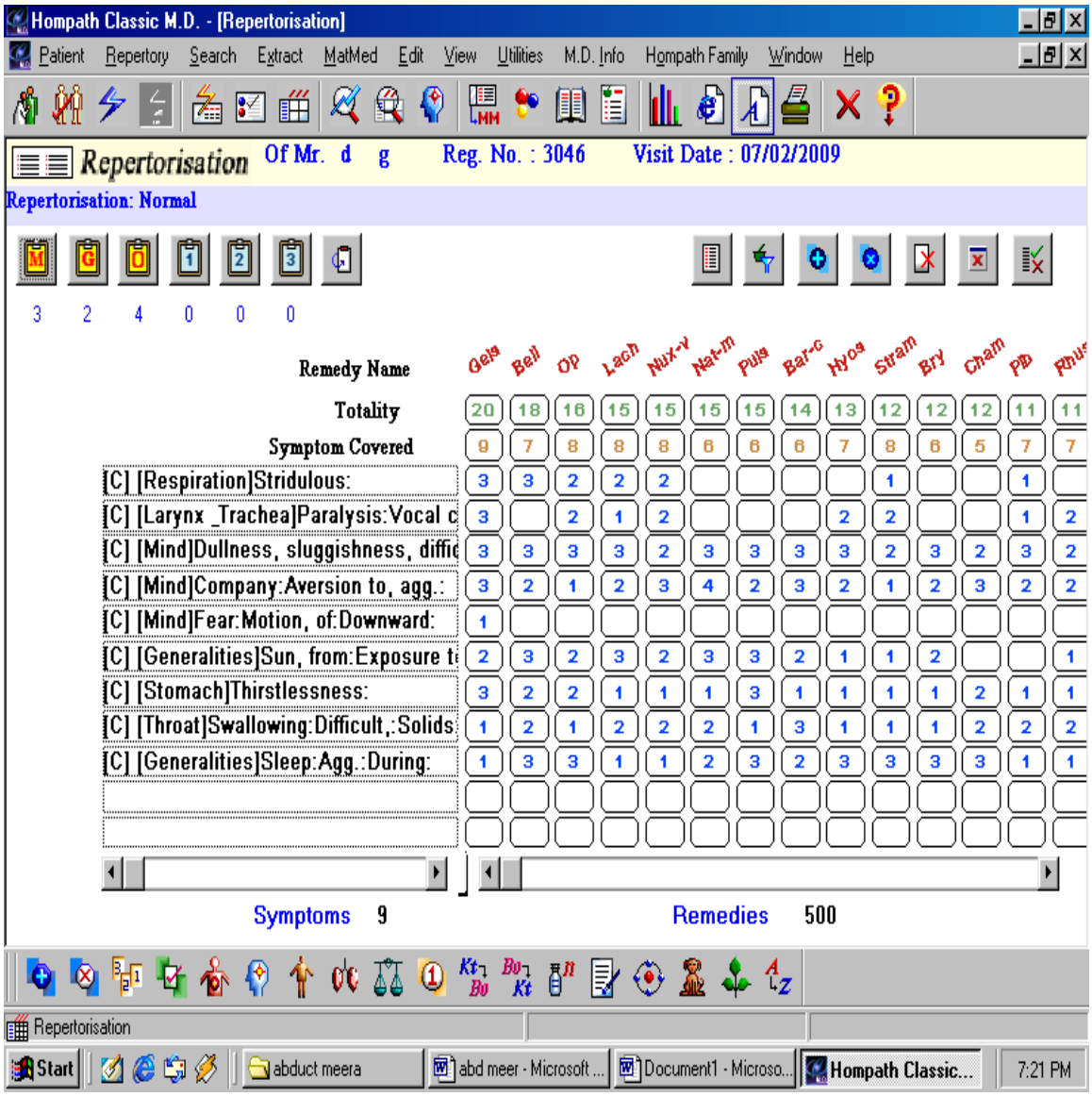
A 8 years male child reported with respiratory stridor since 6 months which was pronounced more during sleep and cold weather, resorted to allopathic treatment but there was no relief. ENT surgeon diagnosed it to be case of " Bilateral Abductor Paralysis". Patient had twice an attack of sunstroke in last summer. Although there was bilateral abductor paralysis but patient had no hoarseness of voice. He was very sluggish in his action. Parents reported he does not mix with his fellow friends. On enquiry it was ascertained if he goes to mela or any festivals in the village, he does not take a ride as he has fear of downward motion. Patient was ambithermic, Appetite was normal. He was taking 2/3 glasses of water per day with moist mouth. Stool was normal, Urine was normal. He was feeling difficulty in swallowing food but not to drink. Past history of skin disease and took allopathic treatment. Mother suffered from pregnancy induced

hypertension. Father had bronchial asthma. Patient was a student in class III and academic performance was below average.

Totality of symptoms of above case was built up which were as follows:

a. Fear of downward motion
b. Aversion to company
c. Dullness
d. Thirstlessness
e. Stridor < cold / night
f. Bilateral abductor paralysis
g. Dysphagic to solid foods
h. H/O sunstroke

The case was repertorised and results were as follows:



The drug of choice was Gelsemium
Which was prescribed in fifty millesimal potency that is

11th Dec 2005

Rx

Gels- 0/1 0/2 0/3 0/4 (1 Oz 8 dose OD)

Followup-

16th Jan 2006

- Patient had no stridor in day time, stridor was felt only during sleep but not aggravated to cold
- Patient who was ambithermic started feeling warmth to heat of sun, Dysphagic to solid food disappeared. Thirst became normal

Rx

Gels – 0/5 0/6 0/7 0/8 (1 Oz 8 dose OD)

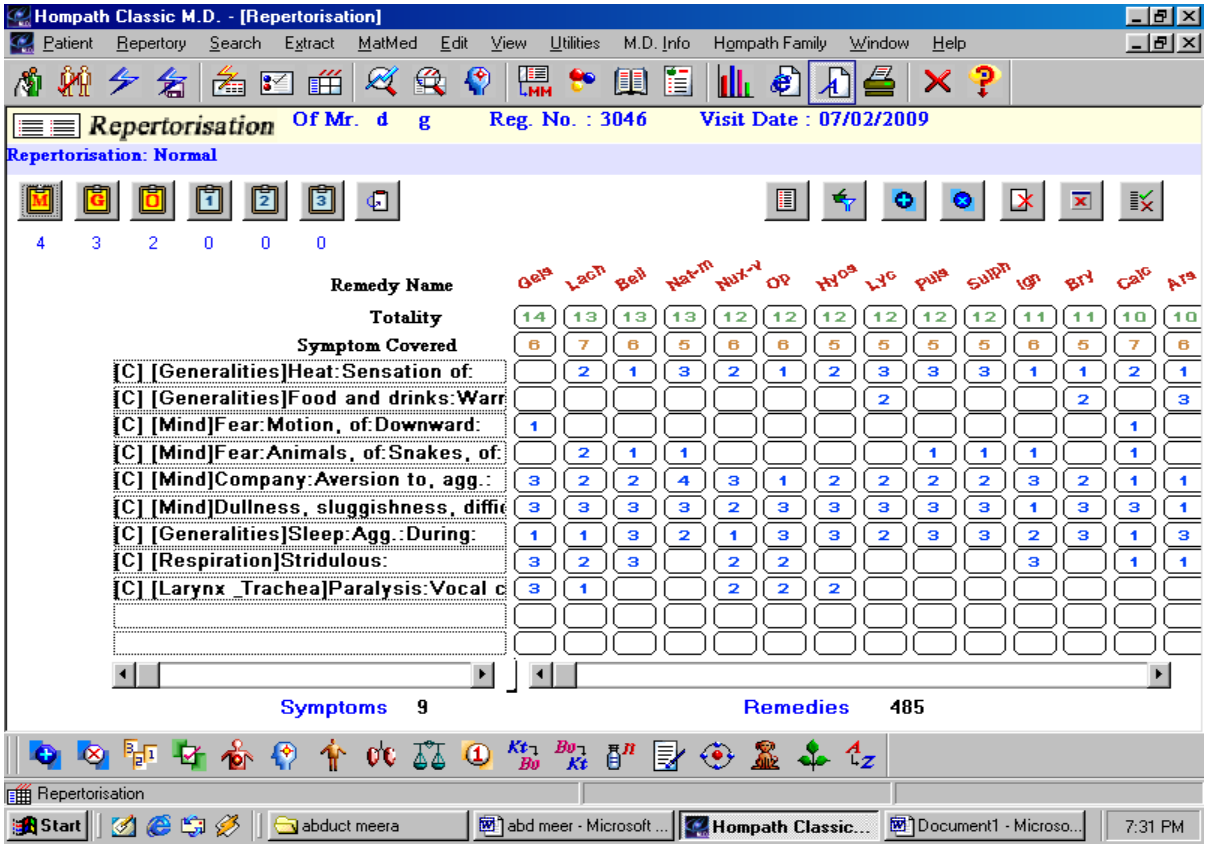
21st Feb 2006

Patient had no much improvement
Condition remained in same state
During review it was ascertained
He is hot patient
Desire for warm food
On further enquiry it was determined that patient is afraid of snakes
He was prosecuting in a public school where use of tie is a customary but mother reported he is not using it.

Totality of symptoms was rescheduled to

1. Hot Patient
2. Desire warm food
3. Fear of snakes
4. Fear of downward motion
5. Company aversion to
6. Dullness
7. Sensitive at neck region
8. Stridor < sleep during
9. Paralysis of vocal cord

Above symptoms were repertorised and results obtained are presented below



25th March 2006

Rx

Lachesis 0/1 0/2 0/3 0/4 (1 Oz 8doses OD)

28th April 2006

-No stridor
-Patient was well
-Patient was followed for 4 more months and there was no other symptoms and declared to be cured.

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