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Neuropathologic chewing in comatose children

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Abstract

This paper reviews the etiology and management of neuropathologic chewing in 16 comatose children. These patients exhibited ruminant chewing and bruxism that often resulted in self-inflicted oral soft tissue trauma. The success of using various dental appliances was found to be associated with the neurologic status of these patients. Variables such as etiology, depth and duration of coma, and age of patient can influence the medical prognosis and hence the success of using these appliances. In cases where the use of dental appliances were not successful, other managerial techniques are discussed.

Patients in coma, from whatever cause, may develop chewing movements presumably similar to those seen in healthy people during sleep. If coma is prolonged, these chewing movements may be so powerful and persistent as to cause self-inflicted trauma to the soft tissues of the oral cavity (Fig 1) and attrition of the teeth.[1] Guyton has termed this condition neuropathologic chewing.[2]

The literature concerning neuropathologic chewing is scarce. The exact mechanism of the coordination of tongue and jaw movements during mastication is still unclear. Hanson et al. designed an intraoral dental appliance based on the neurophysiology of jaw movement in comatose patients.[3] Others fabricated tongue stents[4] and removable appliances[5] for adults to prevent self-inflected trauma to the tongue. Freedman et al. reported success in managing a 2-year-old patient with neuropathologic chewing with a removable bite block.[6] Peters et al. reported success in managing an 8-year-old patient with a tongue stent and circummandibular wires.[7]

Dental services in hospitals frequently are called upon to assist in the management of comatose patients who exhibit bruxism. Some of these cases are complicated by severe intraoral soft tissue laceration as a result of the uncoordinated movement of the tongue and jaw. Thus, it is desirable for dentists to understand this condition and develop effective methods for managing these patients in a hospital setting.

A retrospective study was made on a series of 16 comatose patients with neuropathologic chewing who were referred to the dental service of the Children's Hospital of Philadelphia between 1979 and 1984. Appliances were made for 10 of these patients. The criteria for fabrication of an appliance and the success of using different appliances are discussed in this paper. A standard methodology is proposed for managing hospitalized patients with neuropathologic chewing.

The Comatose Patient

Coma is a symptom closely related to the integration of functions of the cerebral cortex and the reticular activating system. It can be caused by any agent chemical, physical, or biological that is capable of interrupting the integrity of the arousal system. Functionally, the state of coma originates either from brain stem injury (e.g., herniation) or diffuse cortical injury, such as toxicity, anoxia, or metabolic imbalance. The various causes can be classified into supratentorial, subtentorial, or metabolic.[8]

Evidence suggests that meningitis, encephalitis, hypoxia, epilepsy, and metabolic insults are the most common causes of coma in children.[9] The outcome of coma is either recovery, which can be complete or incomplete, or brain death. If recovery is incomplete, the remaining disability can be termed moderate, severe, or a form of vegetative state. The outcome of nontraumatic coma is more favorable in the child than it is for the adult, with almost 60% of patients either recovering completely or having only a mild handicap.[9]

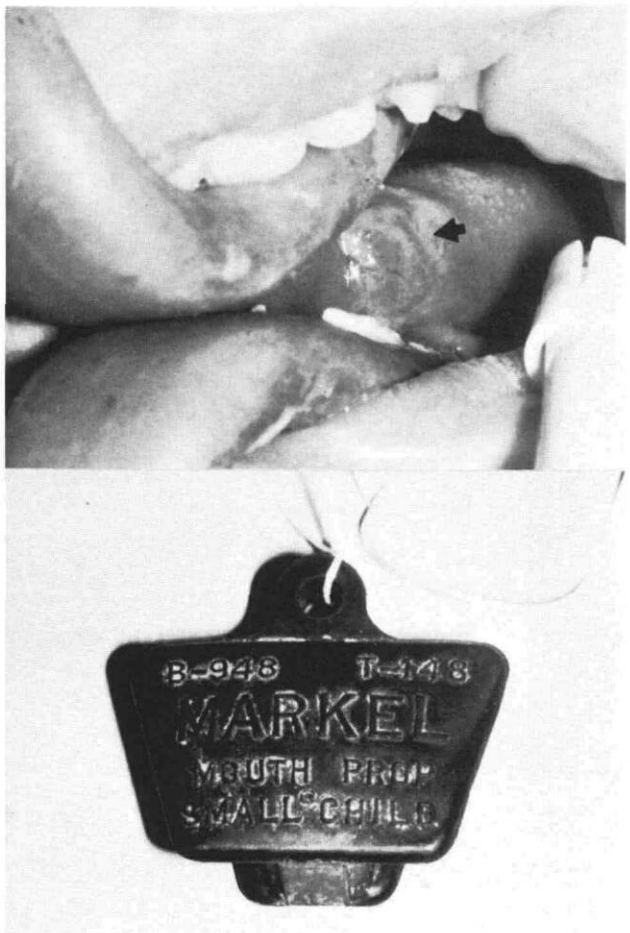

FIG 1. (*top*) Typical self-inflicted trauma in a comatose patient. Immediate relief was achieved with a ratchet (Molt) mouth prop.

FIG 2. (*bottom*) The McKesson rubber bite-block is hygienic and easy to insert.

Neurophysiology

Masticatory movements are normally rhythmic and automatic. The learning of these movements is associated with the cerebral cortex, the reticular formation, and the extrapyramidal system.[2] Jaw openings and closings are coordinated by neurons of the trigeminal sensorimotor complex in the brain stem referred to as the pattern generator.[10] The central programming of this generator may be modified by higher centers in the brain as well as peripheral influences.

Coordination of tongue and jaw movements is necessary during normal mastication and likely to be elaborated by the same generator,[11] which is why the tongue or cheek muscles usually are not bitten during routine movements of the mouth, chewing, speaking, or swallowing.

Patients in coma may have sustained injuries to the cerebral cortex, the reticular or pyramidal system, the trigeminal nucleus, or the hypothalamus, and the fine coordinated movements of the jaw and tongue may have been lost. Jackson states that the placement of a bolus between the teeth perpetuates myotactic masticatory reflex.[4] In comatose patients, the tongue can become juxtapositioned between the teeth resulting in this chewing reflex.

Management of Comatose Children with Neuropathologic Chewing

Several important factors need to be considered before initiating dental treatment for a comatose patient. A careful history should be taken which would include the etiology of coma (Table 1), the age of the patient, the depth and duration of coma, and how rapidly the patient was resuscitated in instances due to anoxia. These are all important factors in predicting the outcome of the comatose patient. The design of any dental appliance also depends on the prognosis of the patient, the neurologic status, and the severity of chewing and/or bruxing.

When examining the patient, dentists should be familiar with the clinical findings in stupor and coma[9] (Table 2). A rapid neurologic assessment should be performed to establish the severity of injuries to the brain.[12] The rapid neurologic assessment is a systematic way to examine the airway, motor functions, and cranial nerves of patients who encounter injuries to the head and neck. The protocol is complete and yet simple enough to be performed by dental practition-

TABLE 1. Common Etiologies of Unconsciousness

Supratentorial
Trauma
Cerebral vascular accident
Cerebral venous thrombosis
Subdural empyema
Intracerebral tumor
Subtentorial
Tumors
Trauma
Primary hemorrhage
Metabolic
Anoxia
Hypoglycemia
Diabetic ketoacidosis
Electrolyte abnormalities
Exogenous poisonings
Meningitis
Encephalitis
Reye's syndrome
Hepatic encephalopathy

From Packer RJ, Berman PM: Coma, in Textbook of Pediatric Emergency Medicine, Fleischer G, Ludwig S, eds. Baltimore; The Williams and Wilkins Co, 1983.

Table 2. Clinical Findings in Stupor and Coma

Level of Damage or Depression	*Respirations*	*Pupils*	*Oculovestibular (caloric) Responses*	*Motor Response*
None (psychogenic unresponsiveness	Eupnea or hyperventilation	2–3 mm, reactive	Nystagmus bilaterally	Voluntary or absent
Cerebral hemisphere	Sighing, eupnea, Cheyne-Stokes (CSR)	2 mm, reactive	Tonic conjugate deviation	Paratonia, paretic
Diencephalon	Eupnea or CSR	1.5–2 mm, reactive	Tonic conjugate deviation	Paretic or decorticate
Uncal herniation	Eupnea or central neurogenic hyperventialation (CNH)	Ipsilateral dilation	Incipient III nerve weakness	Ipsilateral or contralateral parasis, paratonia
Midbrain	CSR or CNH	Midposition 4–6 mm, fixed, irregular	Sluggish or suggestive of internuclear ophthalmoplegia	Decorticate or decerebrate
Pons	Apneustic, cluster or ataxic	Pinpoint, often temporarily fixed	Absent	Decerebrate or lower extremity flexor response
Medulla	Ataxic or apnea	Small (2 mm), reactive	Unaffected	Flaccid or lower extremity flexor response

From Plum F, in Scientific Foundations of Neurology, Critchley M, O'Leary JL, Jennett B, eds. Philadelphia; Davis Co, 1972.

ers in their offices. Teasdale and Jennett developed the Glasgow Coma Scale that correlates the level of consciousness and severity of injury to the head.[13] The neurosurgeon then should be consulted to learn the neurologic status and prognosis of the patient. An oral examination should be performed to detect the presence of ruminatory movements of the jaw, bruxism, and the severity of self-inflicted trauma to intraoral soft tissues. The anesthesia team should be consulted as to the risk of sedating and/or paralyzing the patient if impressions must be obtained for the fabrication of dental appliances.

Indications for the need of fabricating dental appliances should be based on the following criteria:

1. The presence of persistent ruminatory movements of the jaw or bruxism which is usually of more than 24-hr duration
2. The presence of intraoral soft tissue lacerations.

If either of the above criteria is absent, the fabrication of appliances is not immediately necessary.

Numerous methods had been tried to prevent self-inflicted trauma by comatose patients including acrylic bite-blocks, ratchet mouth props, padded tongue blades, and intermaxillary fixation. The use of any dental appliance should be based on the neurophysiology of neuropathologic chewing with modifications based on the special needs of a particular patient.

Patients who had mild ruminatory movements treated by the authors responded well to a taped mouth prop[a] or a rubber bite-block[b] (Fig 2). These are hygienic appliances, easy to insert, and no laboratory preparation is necessary. These appliances also are indicated in cases where immediate relief is needed and can be used temporarily until further treatment decisions are made.

The removable custom-made appliance with a posterior bite-block is the next most hygienic appliance for patients with mild ruminatory movements (Fig 3). The detailed fabrication of this appliance is well described and allows tongue movement, but prevents the tongue from acting as a bolus which causes reflex chewing patterns.[6] The amount of mandibular opening is controlled by the height of the posterior bite-blocks. Retention is usually adequate if most of the primary or permanent dentition is present. The disadvantages of using this appliance are the necessity of paralyzing the patient in order to obtain the impression and the extensive laboratory work required.

In patients with powerful chewing movements, the use of removable appliances usually fails because of problems of retention. A fixed tongue depressing stent may be necessary to temporarily depress the tongue and prevent it from being traumatized.[7]

Another method of preventing self-inflicted oral

[a] Molt mouth prop — Hu Friedy: Chicago, IL.
[b] McKesson rubber bite-block — Silverman: Plymouth Meeting, PA.

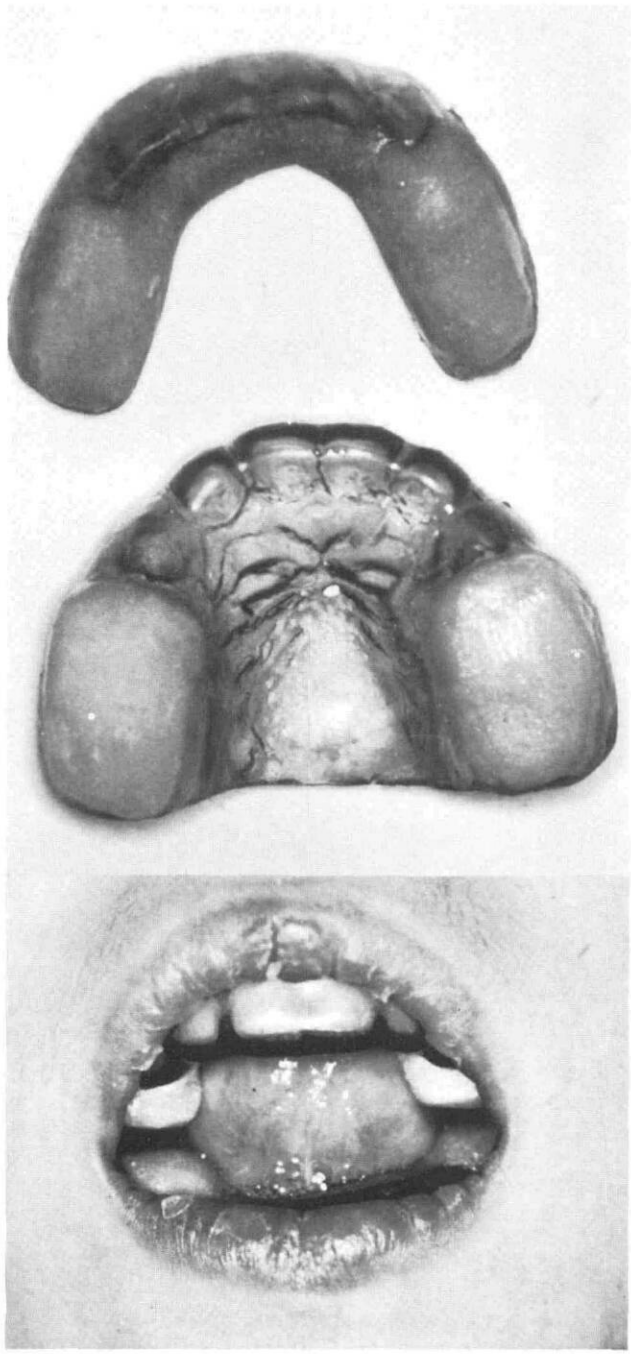

FIG 3. The removable custom appliance with a posterior bite-block allows free range of movements but prevents the tongue from acting as a bolus. This appliance requires laboratory fabrication.

trauma is the fabrication of a soft acrylic custom tray (Fig 4). The custom tray can be fabricated by molding vinyl mouthguard material[c] on the impression models using the dental molding machine. This appliance does not control the height of the posterior opening

[c] Buffalo Dental Mfg Co, Inc: Syosett, NY.

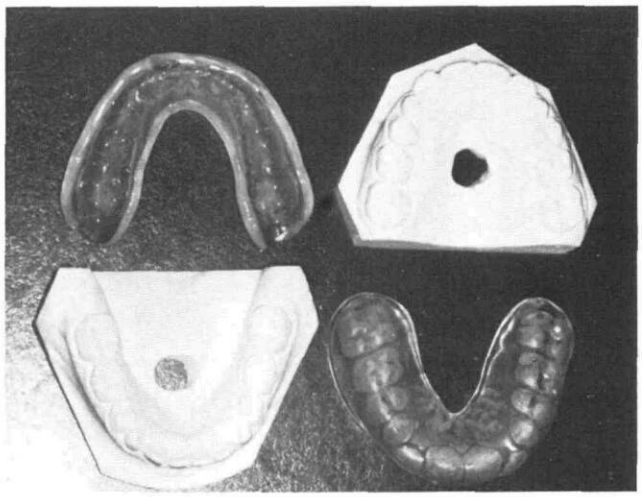

FIG 4. The soft acrylic custom tray is easy to insert but does not control the height of the posterior opening.

and is not as rigid an appliance as the posterior bite-block, but it is easy to fabricate and insert and does not require extensive laboratory work.

The authors do not recommend intermaxillary fixation because it is very difficult to maintain good oral hygiene and removable appliances are eventually necessary if and when the fixation is removed.

Finally, for patients who have mild chewing movements with no apparent intraoral damage, fabrication of appliances should be delayed since in some cases neuropathologic chewing may be a self-limiting phenomenon. These patients are observed daily for any signs of intraoral traumatic injuries and if necessary an appliance is fabricated and employed.

A Retrospective Study of Sixteen Comatose Children

A retrospective study of 16 pediatric comatose patients with neuropathologic chewing was carried out (Table 3). These patients ranged in age from 11 months to 16 years and were comatose due to a variety of causes including anoxia, metabolic disorders, meningoencephalitis, multiorgan failure, and hypogammaglobulinemia.

Dental appliances were fabricated for 11 of the patients who met the criteria for appliance fabrication. Eight patients were managed successfully with bite-block therapy. These patients exhibited healing of the lacerated soft tissues. In addition, due to the concomitant improvement in neurologic status, 6 of the 8 patients did not have clenching or chewing movements after wearing the appliance a few months.

Two other patients were treated with soft acrylic custom trays because of a poor recovery prognosis. In 1 patient, the remaining teeth were extracted due

TABLE 3. Retrospective Study of Sixteen Comatose Patients 1979-84

Patient	*Age*	*Etiology*	*Prognosis*	*Treatment*
BB	5	Anoxia	Good	Acrylic bite-block
WK	1.5	Anoxia	Good	Acrylic bite-block
KB	3	Anoxia	Good	Acrylic bite-block
LB	4	Hypogammaglobulinemia	Poor	Acrylic bite-block
JG	4	Trauma	Fair	Acrylic bite-block
CW	4	Anoxia encephalopathy	Good	McKesson bite-block
KK	2	Anoxia encephalopathy	Fair	McKesson bite-block
SC	4	Anoxia	Poor	McKesson bite-block
CE	16	Multiorgan failure	Poor*	Molt mouthprop
RB	13	Metabolic	Poor*	Custom tray
MT	2.5	Meningoencephalitis	Poor*	Custom tray-Extraction
AJ	1	Meningitis	Good	Follow-up observation
AS	3	Anoxia	Good	Follow-up observation
RC	9	Anoxia	Fair	Follow-up observation
CS	2	Anoxia	Fair	Follow-up observation
FA	2.5	Cardiac failure	Fair	Follow-up observation

* Patient expired during treatment.

to poor retention of the appliance and powerful jaw movements. These 2 patients exhibited soft tissue improvement, but died within a short period after initiating treatment. In 1 other patient, due to the limited mouth opening from TMJ disease, treatment was limited to the use of a mouth prop. One patient (LB) was diagnosed as having end-stage poliomyelitis with progressive neurologic deterioration. At age 4 she showed evidence of neuropathologic chewing. Due to her poor prognosis and in anticipation of further deterioration of neurologic status, a bite-block was fabricated to prevent further trauma to the intraoral soft tissues. The patient tolerated the appliance well under parental supervision until she died from respiratory distress.

In the other group of 5 patients for which dental appliances were not indicated, dental treatment included observation alone. In 4 of the cases, neuropathologic chewing was self-limiting. One patient continued to have a bruxing and chewing habit but without associated intraoral damage.

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