

Educational Module 5

Dysphagia and Nutrition Post ABI

5. Dysphagia

Dysphagia is defined as difficulty or discomfort with swallowing. Morgan and Ward ¹ have noted that traumatic brain injury, associated with focal and diffuse cortical and brainstem damage, may impair swallowing ability and lead to the development of dysphagia and aspiration. Swallowing has four sequential coordinated phases which are summarized in Table 5.1.

5.1 Normal Phases of Swallowing

Q1. Describe the 4 phases of normal swallowing

Answers

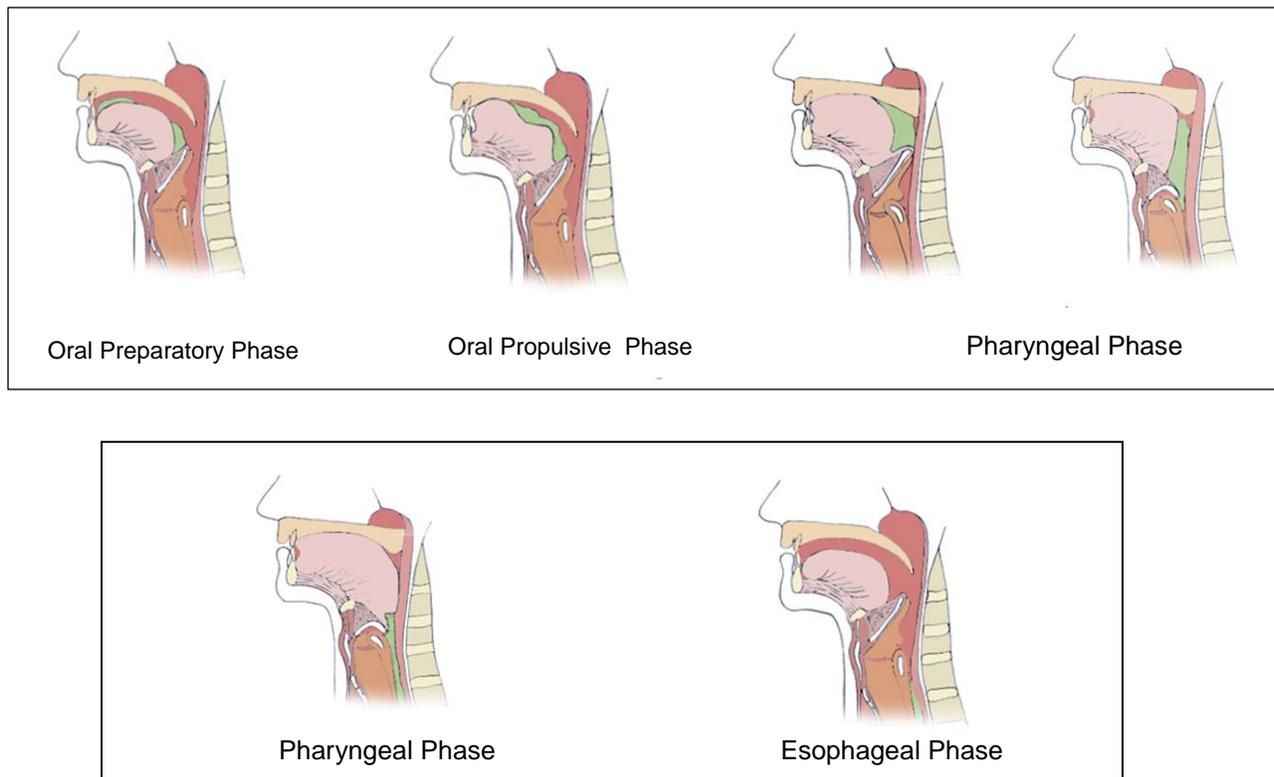
1. Oral preparatory phase
2. Oral propulsive phase
3. Pharyngeal phase
4. Esophageal phase

Discussion

Table 5.1: The 4 Phases of Normal Swallowing².

Phase	Characteristics
Oral Preparatory Phase	Food in the oral cavity is manipulated, masticated and mixed with saliva in preparation for swallowing. The back of the tongue controls the position of the food, preventing it from falling into the pharynx.
Oral Propulsive Phase	The tongue transfers the bolus of food to the pharynx, triggering the pharyngeal swallow.
Pharyngeal Phase	Complex and coordinated movements of the tongue and pharyngeal structures propel the bolus into the esophagus, while protecting the airway.
Esophageal Phase	Coordinated contractions of the muscles of the esophagus move the bolus through the esophagus towards the stomach.

Figure 1: The phases of swallowing



5.2 Pathophysiology of Dysphagia

Q2. What is the pathophysiology of dysphagia following ABI?

Answer

1. Pharyngeal muscle dysfunction and incoordination due to loss of CNS control.

Discussion

Dysphagia post-ABI has been attributed to pharyngeal muscular dysfunction and lack of coordination secondary to central nervous system loss of control. The most common swallowing problems among patients with ABI included, prolonged oral transit (87.5%), delayed swallow reflex (87.5%), valleculae pooling (62.5%), dysphagic and pyriform sinus pooling (62.5%). Aspiration reportedly occurred in 37.5% of patients with dysphagia³.

5.3 Incidence and Natural History of Dysphagia Post ABI

Q3. What is the incidence of dysphagia post ABI?

Answers

1. Incidence of dysphagia entering rehabilitation post-ABI ranges from 42-65%.

Discussion

The reported incidence of dysphagia in the brain-injured population varies considerably, due in part to differences in the timing and method of assessment and the initial level of severity. Among patients admitted to a rehabilitation facility Mackay et al. ⁴ estimated the incidence of dysphagia ranged from 25-42%, which is similar to Cherney and Halper ⁵ who reported the incidence of swallowing disorders in an acute rehabilitation setting between 27% to 42%. Among patients 12 to 45 years, Field and Weiss ³ reported a 30% incidence of dysphagia in patients with head injuries admitted to a rehabilitation facility. Ward and Morgan ⁶ have noted that studies reporting on dysphagia frequency in TBI patients in rehabilitation settings vary from 25-78%; the most recent studies report an incidence of 42-65%.

5.4 Aspiration Post ABI

Q4. Define aspiration

Answer

1. Entry of material into the airway below the level of the true vocal cords

Discussion

Aspiration is defined as “entry of material into the airway below the level of the true vocal cords”. Since many patients with dysphagia do not aspirate, the two terms are not synonymous, although they are closely associated.

5.4.1 Incidence of Aspiration Post ABI

Q5. What is the incidence of aspiration post ABI?

Answer

The incidence of dysphagia in patients entering rehabilitation post-ABI ranges from 25-78%. This incidence has been shown to vary depending on the definition of dysphagia used and the acuity of the patient at admission. An incidence of 42-65% in patients admitted to an ABI rehabilitation unit has been observed in more recent studies.

Discussion

Terre and Mearin⁷ evaluated aspiration improvements in 26 patients at 1, 3, 6, and 12 months post ABI. Videofluoroscopic (VFS) results indicate that aspiration decreased for the majority of patients during the 12 month period following their injury. For the majority of patients the most significant changes were seen at the 3 month evaluation period.

O’Neil-Pirozzi et al.⁸ studied 12 patients all of whom were trached. Patients successfully completed a modified barium swallow (VMBS). Only 3 patients aspirated on some of the various liquids introduced to the patients. These three patients were either in a minimally responsive

state or a vegetative state at the time of testing. All patients were given various oral exercises, or taste and thermal stimulation to improve swallowing.

Mackay et al.⁹ performed a series of VMBS studies on 54 young severely brain injured patients, an average of 17.6 days post-injury and noted a 61% incidence of dysphagia. Of these patients 41% aspirated. Other swallowing abnormalities included loss of bolus control (79%), reduced lingual control (79%), and decreased tongue base retraction (61%) delayed trigger of swallowing reflex (48%), reduced laryngeal closure (45%), reduced laryngeal elevation (36%), unilateral pharyngeal paralysis (24%), absent swallow reflex (6%) and cricopharyngeal dysfunction (3%)⁹.

Schurr et al.¹⁰ conducted bedside evaluations in 47 patients. Of these, 31 were admitted to the VMBS study. VMBS results indicate that 22 of the 31 patients aspirated during feeding. Five patients had laryngeal penetration and aspiration was observed in another 8. All responded to a modified diet.

5.4.2 Silent Aspiration

Q6. Define silent aspiration. How common is it following ABI?

Answer

1. Penetration of food below the level of the true vocal cords, without cough or any outward sign of difficulty.
2. The incidence of silent aspiration in ABI patients has not been well documented.
3. Such cases may be missed in the absence of VMBS studies.

Discussion

Aspiration cannot always be diagnosed by a bedside examination. Patients may aspirate without outward signs. **“Silent aspiration”, is defined as “penetration of food below the level of the true vocal cords, without cough or any outward sign of difficulty”**¹¹. Detailed clinical swallowing assessments were shown to under diagnose or to miss cases of aspiration¹²⁻¹⁴. Silent aspirators were considered to be at increased risk of developing more serious complications such as pneumonia. **Silent aspiration should be suspected in the ABI patient with recurrent lower respiratory infections, chronic congestion, low-grade fever or leukocytosis**¹⁵. **Clinical markers of silent aspiration may include a weak voice or cough or a wet-hoarse vocal quality after swallowing.** Lazarus and Logemann¹⁶ identified aspiration in 38% of their TBI patient group, noting many of these patients, despite aspirating, did not produce a reflexive cough and they required prompting to clear aspirated material⁶. In a more recent study by Terre and Mearin⁷, they found approximately 33% of their subjects were silent aspirators. Dietary changes were made to reduce the risk of aspirating. For many, issues with aspiration seemed to resolve within the 12 months of the study.

5.5 Assessment of Dysphagia Post ABI

Q7. Describe an Approach to the Assessment of Dysphagia Post ABI.

Answer

1. Screening involves a bedside clinical evaluation to determine whether the patient has dysphagia or not.
2. Assessment describes the dysphagia in detail, determines the severity of the problem, and guides a management approach..
3. Patients are initially kept NPO.
4. Clinical assessment is initially performed and if necessary a videofluoroscopic modified barium swallow (VMBS) is done.

Discussion

A thorough assessment of swallowing function involves both a bedside clinical evaluation plus a radiological procedure and will help to guide appropriate intervention strategies. The clinical bedside examination usually involves general observations, an oral motor examination, and a swallowing/feeding trial ⁶ including the introduction of one or several teaspoons of water and in some protocols, various consistencies of food and liquids. In one protocol, if patients were unable to successfully swallow a minimal amount of fluid, a small cup of water was carefully introduced. The full assessment is described elsewhere ¹⁷.

Although many of the tools used in practice to assess swallowing disorders in those who have sustained an ABI, none of these tools have been studied extensively or specifically within this population.

Swallowing Recommendations (ABIKUS Guidelines 2007 ¹⁸)

Persons with moderate to severe ABI should:

- *Be screened for risk of dysphagia and aspiration by an appropriately trained clinician*
- *Be assessed by a speech language pathologist if there are features of dysphagia or aspiration to determine the appropriate feeding strategy*

(ABIKUS A) (G69-p.27)

5.5.1 The Bedside Clinical Examination

Q8. Which bedside clinical examinations have been shown to be the most useful?

Answer

1. Only two tests, the abnormal pharyngeal sensation and the 50 ml water-swallowing test have sufficient evidence supporting their use.

Discussion

Several forms of clinical or bedside swallowing evaluations have been described for the purposes of screening and/or assessment. Some of these methods target specific functions or tasks, while others evaluate swallowing ability using a more comprehensive approach. These methods may or may not include a water-swallowing test. While bedside assessment is non-invasive and easy to perform, this method has been shown to predict poorly the presence of silent aspiration. Smith et al. ¹⁹ reported that aspiration cannot be distinguished from laryngeal

penetration using a bedside evaluation, resulting in the over diagnosis of aspiration and, in some cases, needless dietary restrictions.

The results of a recent systematic review by Martino et al.²⁰ evaluating the screening accuracy of 49 individual clinical screening tests for oropharyngeal dysphagia suggest that there is only sufficient evidence to support the value of two tests: abnormal pharyngeal sensation and the 50 mL water-swallowing test. Both of these tests assessed only for the presence or absence of aspiration. Their associated likelihood ratios were 5.7 (95% CI 2.5-12.9) and 2.5 (95% CI 1.7-3.7), respectively. Limited evidence for screening benefits suggested a reduction in pneumonia, length of hospital stay, personnel costs and patients.

5.5.2 Water Swallowing Test

The water-swallowing test although not specifically studied in ABI, warrants inclusion here. This test has however been studied extensively within the stroke population, to establish its validity as part of a clinical swallowing assessment. While the original test required a patient to swallow 3 oz (90ml) of water, smaller amounts have also been used.

Discussion

When not provided, the positive predictive value (PPV), negative predictive value (NPV) and positive and negative likelihood ratios (+ LR & -LR) for the water-swallowing test were calculated for each study and summarized in the following table. The gold standard used to confirm aspiration was either VMBS or FEES examination. (We used the data provided by the authors from DePippo et al.²¹ and found that the sensitivity and specificity reported were actually the PPV and NPV). A likelihood ratio (LR) of either greater than 10 or less than 0.1 is considered to be strong evidence to either rule in or rule out disease (in this case, the presence of aspiration), while LRs less than 2 are considered to be small.

Table 2: The PPV, NPV, + LR & - LR for the detection of Aspiration Using the Water Swallowing Test

Study	PPV (%)	NPV (%)	+ LR	- LR
DePippo et al., ²¹	59	76	1.75	0.37
Garon et al., ²²	79	54	3.24	0.24
Chong et al., ²³	81.8	58.8	2.12	0.33
Lim et al., ²⁴	78.6	81.8	3.39	0.25
Wu et al., ²⁵	78.6	73.3	5.74	0.57

Two studies authored by Lim et al.²⁴ and Chong et al.²³ combined the results of the water swallowing test and the oxygen saturation test to create a “clinical” or “bedside” assessment tool, increasing the accuracy of the diagnosis of aspiration. The PPV increased to 3.43 in the study by Lim et al.²⁴ and to 2.51 in the Chong et al.²³ study.

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5.5.4 Risk Factors for Dysphagia Post ABI

Q9. What are the risk factors for dysphagia post ABI?

Answers

1. Extent of brain injury
2. Duration of coma ¹⁶
3. Lower Glasgow Coma Score on admission (GCS 3-5) ⁹
4. Severity of CT Scan findings⁴
5. Duration of mechanical ventilation ⁴
6. Tracheostomy
7. Translaryngeal (endotracheal) intubation
8. Severe cognitive and cognition disorders
9. Physical damage to oral, pharyngeal, laryngeal and esophageal structures
10. Oral and pharyngeal sensory difficulties

Discussion

Ward and Morgan⁶ have identified a body of research which has attempted to define those factors which may affect the presence and severity of dysphagia post-TBI ^{4;5;9;26;27}. Injuries that result from translaryngeal intubation or tracheostomy may contribute to swallowing dysfunction in TBI patients²⁷. Morgan and Mackay²⁷ also note that severe TBI patients with neurogenic dysphagia and a tracheostomy, are at particularly high-risk of aspiration. The negative effects can be minimized by ensuring the use of appropriately sized tracheostomy tubes and by avoiding overinflation of the cuff ²⁸. Passy-Muir (Positive Closure) Speaking Valves operated in the closed position can improve voice quality and speech production, while at the same time improve swallowing and reduce aspiration (Passy-Muir Clinical Inservice Outline ²⁹).

5.5.5 Diagnosis of Aspiration Post ABI

Q10. What are the risk factors for aspiration post ABI?

Answers

The risk of dysphagia related aspiration is proportional to the initial severity of the head injury. A history of a tracheostomy or mechanical ventilation may also be associated with increased risk of aspiration. A summary of all risk factors is listed below:

1. Lower Glasgow Coma Score (3-5) ⁴
2. Presence of a tracheostomy
3. Poor cognitive functioning
4. Hypoactive gag reflex
5. Prolonged period of mechanical ventilation ⁴
6. Reduced pharyngeal sensation
7. Brainstem involvement
8. Difficulty swallowing oral secretions
9. Coughing/throat clearing or wet, gurgly voice quality after swallowing water
10. Choking more than once while drinking 50 ml of water
11. Weak voice and cough
12. Wet-hoarse voice quality
13. Recurrent lower respiratory infections

14. Low-grade fever or leukocytosis
15. Auscultatory evidence of lower lobe congestion
16. Immunocompromised state

Discussion

Aspiration should be suspected when the ABI patient has any of the following: a complaint of trouble swallowing, an abnormal chest x-ray, congested vocal quality, or a delay in voluntary initiation of the swallow reflex and coughing during or after swallowing¹³. While all ABI patients are potential aspirators there are certain identifiable risk factors that have been recognized as greatly increasing the likelihood of aspiration. Initial severity of brain injury appears to be the strongest predictor of dysphagia related aspiration. Certain risk factors place patients at higher risk for aspiration. Initial severity of brain injury appears to be the strongest predictor of dysphagia related aspiration. Additional factors which may also be strongly related to the severity of injury include the presence of a tracheostomy and the need for mechanical ventilatory support.

5.5.6 Videofluoroscopic Modified Barium Swallow (VMBS) Studies

Q11. Describe the importance of VMBS studies in the diagnosis of aspiration.

Answers

1. Considered to be the “gold standard” in diagnosis of aspiration.
2. Patients who aspirate over 10% of the test bolus or who have severe oral and/or pharyngeal motility problems are considered at high risk of pneumonia.
3. May reveal the mechanism of the swallowing disorder.

Discussion

When aspiration is suspected, the VMBS study is considered by some to be the “gold standard” in confirming the diagnosis¹⁴. A VMBS study examines the oral and pharyngeal phases of swallowing. However, the patient must have sufficient cognitive and physical skills to undergo testing³⁰. The subject is placed in the sitting position in a chair designed to simulate the ideal/optimal mealtime posture. Radio-opaque materials of various consistencies are tested: barium impregnated thin and thick liquids, pudding, bread and cookies are routinely used. Various aspects of oral, laryngeal and pharyngeal involvement are noted during the radiographic examination. The VMBS study can then be followed by a chest x-ray to document any barium, which may have been aspirated into the tracheobronchial tree.

Those patients who aspirate over 10% of the test bolus or who have severe oral and/or pharyngeal motility problems on VMBS testing are considered at high risk for pneumonia^{31:32}. In many cases, it is difficult to practically assess whether 10% or more of the test bolus has been aspirated. Nevertheless, the degree of aspiration seen on VMBS study is a critical determinant of patient management. Predicting whether a patient will develop pneumonia post aspiration is, to some extent, dependent on other factors such as the immune state or general health of the ABI patient.

The VMBS assessment not only establishes the presence and extent of aspiration but may also reveal the mechanism of the swallowing disorder. Aspiration most often results

from a functional disturbance in the pharyngeal phase of swallowing related to reduced laryngeal closure or pharyngeal paresis. A VMBS study is recommended in those cases where the patient is experiencing obvious problems maintaining adequate hydration/nutrition, where concern is expressed regarding frequent choking while eating, or in the case of recurrent respiratory infections. Other factors such as cognition, depression, immunocompromization, and underlying lung disease must also be considered.

Definitive criteria for determining indication for VMBS study have yet to be determined empirically. If a VMBS study is indicated and the result is positive, a second VMBS study may be appropriate in 1-3 months, if swallowing concerns persist.

5.5.6 Blue Dye Assessment for Swallowing

The blue dye assessment for swallowing has been used since the early '70's with patients who have a tracheotomy; however the accuracy of the test has been questioned since the 1980s⁸. **For patients with a tracheostomy, this assessment involves placing blue dye on the tongue or, in the case of the modified blue dye test, mixing it with water or semisolid food.** If blue dye appears in or around the tracheostomy tube, or at defined intervals during suctioning then the patient has possibly aspirated. This test tends to be relatively easy to administer, is inexpensive and it may be performed at a patient's bedside; however, research has shown the test may have a 50% false-negative error rate in the detection of aspirated material³³⁻³⁵. Belafsky et al.³⁵ in a study of 30 patients with tracheostomies conclude that the use of the MEBD test is beneficial with patients who have a tracheostomy tube (82% sensitivity) and in particular those who receive mechanical ventilation (100% sensitivity).

Brady et al.³⁴ in a study looking at the effectiveness of the modified Evans blue dye test (MEBD) and the videofluoroscopic swallow (VMBS) found the MEBD was not able to detect "trace amounts" of aspiration in patients who had a tracheostomy. On the other hand, if patients aspirated more than "trace amounts", then the MEBD was able to detect it. Brady et al. (1999) recommended that MEBD be followed by a VMBS to rule out the possibility of trace aspiration. Although this test is used in practice with those who sustain ABIs, no studies were found looking at its effectiveness with this population specifically. Similar findings were reported by Donzelli et al.³³ and O'Neil-Pirozzi et al.⁸. The Donzelli et al.³³ results indicate the MEBD test was unable to detect aspiration in trace amounts, thus confirming the 50% false-negative error rate. O'Neil-Pirozzi et al.⁸ found the blue dye test was unable to correctly identify aspiration in 20% of study's tracheostomy patients and 38% of tracheostomy patients who were not aspirating.

5.6 Management of Dysphagia Post ABI

Q12. Describe a dysphagia management program at the time of acute care admission in an ABI patient suspected of suffering from dysphagia.

Answers

1. Acutely patients should be NPO until swallowing ability has been determined.
2. A trained assessor should screen all acute patients for swallowing difficulties as soon as they are able.
3. A speech and language pathologist should assess all patients who fail swallowing screening and identify the appropriate course of treatment.
4. VMBS is the "gold standard" for diagnosis of aspiration.

5. An individual trained in low-risk feeding strategies should provide feeding assistance or supervision to patients where appropriate.
6. A dietitian should assess the nutrition and hydration status of patients who fail the swallowing screening.

5.6.1 Best Practice Guidelines for Managing Dysphagia

Best practice guidelines for managing dysphagia were developed by a consensus committee of the Heart and Stroke Foundation of Ontario. Although these guidelines were developed for stroke they are applicable to ABI Rehabilitation. These guidelines are summarized below. Similar guidelines have not been developed yet for ABI rehabilitation.

Best Practice Guidelines for Managing Dysphagia Post-Stroke ³⁶

1. Maintain NPO until swallowing status is determined.
2. Regular oral care, with minimum of water to limit build-up of bacteria.
3. Screen for swallowing status once awake and alert by trained team member.
4. Screen for risk factors of poor nutrition early by trained team member.
5. Swallowing assessment by speech language pathologist to:
 - assess ability to swallow
 - determine swallowing complications
 - identify associated factors which may be compromising swallowing and nutrition.
 - recommend appropriate individualized management program including appropriate diet.
 - monitor hydration status.
6. Where appropriate feeding assistance or mealtime supervision by individuals trained in low risk feeding strategies.
7. Assess nutrition and hydration status and needs of those who fail screening; reassess regularly.
8. Education of patient and family with follow-up upon discharge.
9. Consider the wishes and values of the patient and family concerning oral and non-oral nutrition; provide information to allow informed choices.

5.6.2 Low Risk Feeding Strategies

Q13. Describe why low risk feeding strategies are necessary?

Answer

1. Individuals with dysphagia who are fed by someone else have a 20 times greater risk of pneumonia than patients who are able to feed themselves.

Q14. Describe some of these low risk strategies?

Answers

1. Calm eating environment with a minimum of distractions

2. Patient is properly positioned in an upright position with neck slightly flexed
3. Proper oral care
4. Feed at eye level
5. Feed slowly
6. Drink from wide mouth cup or a straw to reduce the neck extending back
7. Ensure swallowing before offering more
8. Properly position the patient and monitor for 30 minutes after each meal.

Discussion

The Heart and Stroke Foundation Dysphagia Guidelines for stroke patients note **that individuals with dysphagia who are fed by someone else have a 20 times greater risk of pneumonia than those patients who are able to feed themselves**³⁷. It is noted that where dysphagia patients are not able to feed themselves independently, hand-over-hand support should be provided at eye-level positioning. If full feeding assistance is required, it needs to be provided using low-risk feeding strategies (see below).

Low Risk Feeding Strategies in Dysphagia Stroke Patients (HSFO Guidelines)

- Ability of feeder to deal with emergencies, such as choking.
- Calm eating environment with a minimum of distractions.
- Patient properly positioned – upright, midline with neck slightly flexed.
- Proper oral care.
- Feed at eye-level.
- Metal teaspoons (no tablespoons or plastic).
- Feed slowly.
- Drink from wide-mouth cup or a straw to reduce neck extending back.
- Ensure swallowing before offering more.
- Properly position and monitor for swallowing problems for at least 30 minutes after each meal.
- Carefully monitor patient's oral intake.

It's of note that in stroke patients, there is Level 4 evidence that individuals with dysphagia should feed themselves to reduce the risk of aspiration. There are no such studies in ABI. For stroke patients who require assistance to feed there is consensus opinion that low-risk feeding strategies by trained personnel should be employed. There are no such consensus statements made for ABI.

5.7 Management of Dysphagia for Patients with ABI

Q15. How is dysphagia post ABI managed?

Answers

1. Patients undergo a careful clinical assessment and where necessary a VMBS is performed
2. Patient is kept NPO until swallowing status can be determined

3. Where the patient is a severe aspirator, a non-oral feeding tube is inserted
4. Where the patient is a mild to moderate aspirator treatment is determined by the findings of the VMBS
5. For these patients compensatory treatment techniques are used.

Q16. Describe compensatory treatment techniques for dysphagia post ABI?

Answers

1. Postural adjustment of the head, neck and body to modify the dimensions of the pharynx and optimize the flow of the bolus
2. Sensory stimulation techniques to improve sensory input
3. Food consistency and viscosity alterations
4. Modifying the volume and rate of food and fluid presentation
5. Use of intraoral prosthetics

Discussion

The careful management of dysphagia is essential for the successful rehabilitation of acute brain injury patients³⁸. **Ward and Morgan⁶ described the use of three distinct types of rehabilitation programs for dysphagic patients following head injury, based on the status of swallowing function at the time of admission³⁹. The nonfeeding program** was designed as a stimulation program for very low-level patients, in order to prepare them for later feeding³⁹. This program includes desensitization techniques, such as stroking, and applying pressure or stretching, to facilitate normal swallowing, sucking and intraoral responses³⁹. The second program, **the facilitation and feeding program**, uses small amounts of puree consistency food to assist normal feeding patterns³⁹. The third program is referred to as a **progressive feeding program**, where specialized techniques were used to help the patient develop swallowing endurance by systematically increasing the amount of oral intake³⁹. This progressive feeding program continued until the patient was able to consume a complete meal within thirty minutes without difficulties³⁹.

For patients who are safe with some form of oral intake, Ward and Morgan⁴⁰ note that **therapeutic strategies utilized in dysphagia management can be divided into two categories: (a) compensatory treatment techniques** and (b) **therapy techniques⁴¹**.

Compensatory treatment techniques do not involve direct treatment of the swallowing disorder. Their purpose is to reduce or eliminate the dysphagic symptoms and risk of aspiration by altering how swallowing occurs^{41,42}. The types of compensatory strategies include: (a) postural adjustment of the head, neck and body to modify the dimensions of the pharynx and improve the flow of the bolus; (b) sensory stimulation techniques used to improve sensory input either prior to or during the swallow; (c) food consistency and viscosity alterations; (d) modifying the volume and rate of food/fluid presentation; (e) use of intraoral prosthetics⁴¹.

Conversely, **therapy techniques are designed to alter the swallow physiology⁴¹**. They include range-of-motion and bolus handling tasks to improve neuromuscular control without actually swallowing. They also include swallowing maneuvers that target specific aspects of the pharyngeal stage of the swallow. It was noted that medical and surgical management

techniques are included in this category ⁴² with these interventions only introduced once trials with more traditional behavioural treatment techniques have proven to be unsuccessful.

Ward and Morgan ⁴⁰ have noted that ***the efficacy of a large majority of treatments for swallowing disorders have not been studied in the ABI rehabilitation population.*** However, many techniques mentioned above have been studied in other adult populations with neurogenic oropharyngeal dysphagia.

5.9 Treatment of Dysphagia Post ABI

Of note several treatments have been found to treat dysphagia. Included among these are: “*vocal fold adduction exercises, range of motion exercises for the lips, tongue, and jaw, and chewing exercises*” (Logemann, 1993); however, many of these exercises although tested within the stroke or other populations have not been tested specifically within the ABI population.

5.9.1 Oral Motor Exercises (OME)

Exercises introduced with those who have developed a swallowing disorder include various oral motor exercises such as range of motion exercises for the tongue and the pharyngeal structures (Logemann, 1998; pg. 206-210). These exercises are designed to improve strength, movement, awareness and muscle coordination when swallowing (Kramer et al., 2007).

To aid in the improvement of oral transit, exercises to assist in tongue elevation and lateralization may be implemented. Here the patient may be asked to perform very specific tongue exercises in an effort to improve speech and swallowing (Logemann, 1998). Individuals may also be asked to participate in tongue resistance exercises (pushing the tongue against a tongue blade or popsicle stick for 1 second) and bolus control exercises (to allow the patient to learn to control or manipulate items placed in the mouth) (Logemann, 1998).

5.9.1.1 Range of Motion Exercises for the Pharyngeal Structures

Airway Entrance:

Here the individual is placed in a seated position and asked to bear down while holding his or her breath. This exercise is not recommended for those with uncontrolled blood pressure (Logemann, 1998). It is recommended this exercise be done 5 to 10 times each day for 5 minutes.

Vocal Fold Adduction Exercises:

To improve vocal quality and reduce the risk of aspiration, individuals are asked to bear down, with one hand against a chair while producing a clear voice. This exercise is performed 5 times. Following this the individual is asked to repeat “ah” 5 times. Again it is recommended that these exercises be repeated 3 times in sequence, 5 to 10 times each day for 5 minutes. If there is no significant improvement in swallowing at the end of one week, individuals may be asked to pull up on the seat of a chair, while sitting in it, and prolong phonation (Logemann, 1998). This exercise is recommended for those individuals with vocal folds that fail to close completely (Kramer et al., 2007).

5.9.2 The Shaker Exercise

In completing the Shaker Exercise, patients are asked to lay flat on the floor or in bed and raise their heads high enough to see their toes. This position is held for one minute then the patient rests for one. The exercise is repeated three times. Following this sequence, the patient lifts their head, looks at their toes, then lowers their head. This, head up - then down, sequence is repeated 30 times. It is recommended that the Shaker Exercise be completed 3 times per day

for a period of 6 weeks. This exercise has been shown to have some success in improving hyolaryngeal movement; however it has not been studied specifically in the ABI population (Logemann, 2008; Shaker et al., 1997; Shaker et al., 2002).

In a study conducted by Shaker et al. (1997), two groups of age matched healthy adults were divided into one of two groups: those in the Shaker exercise group and those in the sham group (controls). All subjects were asked to swallow 5ml liquid barium. Results indicate that for those in the Shaker exercise group (n=19), the upper esophageal sphincter (UES) opening increased from 8.7 mm to 9.8 mm post intervention.

5.9.3 Swallow Maneuvers

During the acute stage of recovery, patients may experience more swallowing difficulties than they do during later rehabilitation. Failing to address and treat swallowing difficulties in the acute stage may lead to compliance issues with recommended diets for safety and possible setbacks with aspiration pneumonia. Such infections may quickly become a barrier to the patient's ability to participate fully in any formal rehabilitation. Post ABI swallowing difficulties are often the result of eating too quickly, taking large bites, cognitive impairments, and decreased swallowing sensitivity (Logemann, 1998).

To address swallowing difficulties, 4 specific maneuvers have been developed, each addressing a specific dysphagia presentation. For patients to be successful with each of these maneuvers they must have the ability to follow direction, they must be alert and be able to exert the physical effort it takes to do the maneuvers correctly (Karamer et al., 2007).

5.9.3.1 Supraglottic Swallow

This maneuver was *“designed to close the airway at the level of the true vocal folds before and during the swallow”* (Logemann, 1998; Logemann et al., 1997). In this maneuver individuals are asked to hold their breath while swallowing and then to cough immediately after the swallow. This maneuver encourages closure of the true vocal cords in an effort to address reduced or delayed vocal fold closure or delayed pharyngeal swallow. The cough portion of this maneuver is meant to eject any objects or residue caught in the laryngeal vestibule.

5.9.3.2 Super-supraglottic Swallow

During this maneuver, individuals are asked to take a breath in and hold it while bearing down hard, swallow while holding this breath and bearing down, then cough immediately after the swallow (Logemann et al., 1997). This procedure is designed *“to close the airway entrance before and during the swallow”* (Logemann, 1998). The Supraglottic Swallow maneuver is used to address reduced closure of the airway entrance (Perlman & Schulze-Delrieu, 1997).

5.9.3.3 Effortful Swallow

This technique was designed to *“increase posterior movement of tongue base”* (Kramer et al., 2007). As the individual swallows they are asked to squeeze hard with all the muscles (throat and neck muscles) they use for swallowing. The maneuver is intended to address reduced posterior movement of the tongue base

5.9.3.4 Mendelsohn Maneuver

The objective of this maneuver is to address decreased laryngeal movement and discoordination of the swallow (Perlman & Schulze-Delrieu, 1997) Improvements in swallowing function are achieved through *“increasing the extent and duration of laryngeal elevation, thus increasing the duration and width of the cricopharyngeal opening”* (Logemann, 1998). Typically, patients are asked to swallow, but as they do so, to hold their Adam's apple up, for 2-3 seconds

then complete the swallow. This exercise may be recommended to be performed several times a day.

5.9.4 Frazier Free Water protocol

To increase fluid consumption and decrease the risk of dehydration, the Frazier Water Protocol, allows patients, who are receiving thickened liquids, to be given regular, thin water between meals. This practice (and later protocol) arose out of complaints from patients that thickened fluids did not quench thirst in the same way regular thin water does. The regular water, in combination with the recommended thickened fluids, works to assist some patients in better meeting their daily hydration needs. Patients who are NPO are often permitted to have water (following screening) and those who have found success using various postural changes are asked to use these postural maneuvers when drinking water. The Frazier Free Water protocol states that “*by policy any patient NPO or on a dysphasic diet may have water*” (Panther, 2005).

5.9.5 Thermal-tactile Stimulation

Thermal stimulation or thermal-tactile stimulation was developed to stimulate the swallowing reflex of patients who are neurologically impaired (Lazzara et al., 1986). The procedure for thermal-tactile stimulation involves having the patient open their mouth and applying a cold laryngeal mirror at the base of the faucial arches. The mirror, while being in contact with the arch, is then rubbed up and down five times. For those patients who have sustained a “trauma” contact will be made on the normal (non-injured) side of the mouth (Logemann, 1998; p212). Pharyngeal swallow is not triggered at the time of stimulation, but its purpose is to heighten the sensitivity for swallowing in the central nervous system. It is hoped that once a patient attempts to swallow the pharyngeal swallow will be triggered more quickly (Logemann, 1998).

In one study 22 patients who had been diagnosed with dysphagia post stroke, were assigned to either the untreated group or the treated group. Those in the untreated groups were asked to swallow 10 consecutive semi-solid boluses, while those in the treated group had a chilled laryngeal mirror applied to the anterior faucial pillars before they swallowed (Rosenbek et al., 1996). Three strokes were applied to the right and left side of the pillars, then the procedure was reversed. An attempt to keep the probe as cold as possible was made. Following the stimulation, patients were asked to swallow a bolus. Results indicated that the duration of stage transition and total swallow duration was reduced following thermal stimulation. These findings suggest that thermal stimulation using a cold probe may modify swallowing duration (Rosenbek et al., 1996). Although this method appears to be effective, no research on its effectiveness or its efficacy within the ABI population appears to have been conducted. Further research is recommended.

5.9.6 Postural Techniques

Moving the patient, changing position of the head, neck and/or body may assist in changing the direction of the bolus flow, thereby reducing the risk of aspiration. There are five postures, chin-up, chin-down, head turn (left or right), head tilted (left or right) and lying down, that have been shown to have some success in assisting individuals improve their swallowing function (Logemann, 2008).

1) Chin down posture:

- a) Helpful for those who have tongue base retraction issues;
- b) Mechanism of change widens the valleculae, allowing the valleculae to contain the bolus in event of pharyngeal delay.

2) Chin up posture:

- a) Helpful for those who have oral tongue propulsion problems;

- b) Aids in gaining adequate lingual pressure to drive the food or liquid out of the mouth and into the pharynx.
- 3) **Head turn:**
 - a) Involves rotating the head to the side that is damaged;
 - b) Bolus is then directed down the “normal” safe side.
- 4) **Head tilt:**
 - a) Head is tilted toward the stronger side, to promote the flow of food and liquid to go down that side.
- 5) **Lying down:**
 - a) Shown effective in those with posterior pharyngeal wall contraction or reduced laryngeal elevation with resulting residue and subsequent aspiration after swallowing.
 - b) Residual or pooling of food or liquid in the pharynx is kept from falling into the airway as gravity pulls the bolus towards the posterior pharyngeal wall and in this way bolus may be more easily moved into the esophagus (Drake et al., 1997; Rasley et al., 1993).

For individuals who have significant cognitive deficits post injury, having the patient engage in any one of these techniques may be challenging. It has been suggested that patients with *“oral and pharyngeal deficits remain upright for 30 minutes post-meal to reduce the risk of aspirating, take controlled bites and sips, alternate solids and liquids, cue the patient to take multiple swallows and train the patient to clear or remove food that is pocketed in the mouth”* (Kramer et al., 2007).

5.9.7 Diet Modification

To date there has not been a "typical" dysphagia diet developed (Logemann, 1989). The consistency of food should be chosen based on the specific nature of the problem. It should also be noted that restrictions to diet and specific consistencies of food should be the last strategy examined (Perlman & Schulze-Delrieu, 1997). Restrictions to diets and consistencies, especially thin fluids, can be very challenging for individuals (Perlman & Schulze-Delrieu, 1997). That said, diets for those who have been identified with dysphagia not remedial to other compensatory strategies are generally determined by speech language pathologists or others trained in dysphagia management. These patients may begin with a very restrictive diet (liquids of various consistencies – purees) and move to less restrictive diets (diced to regular foods) at a pace that has been deemed safe for that individual (Kramer et al., 2007). Asking the patient to limit the amount of food they attempt to swallow (taking smaller bites) will also help reduce difficulties with swallowing.

In practice, there is a great deal of variation in the dysphagic diets available at various hospital/centres/facilities as well as great variation in the names they are given. Although an attempt has been made (McCallum 2003) to standardize dysphagic diets, there continues to be much variation in practice, setting to setting.

5.9.8 Passy-Muir Speaking Valve (PMV)

Aspiration is often problematic in patients who have a tracheostomy. These patients are essentially unable to achieve the apneic interval necessary for an efficient swallow. It is thought that, normalization of subglottic air pressure, achieved through placement of a Passy Muir Speaking Valve (PMV), reduces the potential for aspiration. In fact, in a study by Gross et al. (1994) they found a *“tenfold in subglottic pressure with the speaking valve in place”*.

Many of these patients have been shown to benefit from the PMV, designed by Victor Passy. The valve may be attached to the 15mm connector found on most adult tracheostomy tubes (Dettelbach et al., 1995; Passy et al., 1993). With the PMV in place, a noticeable decrease in

the amount aspirated has been observed. While wearing the valve, patients also have the opportunity to more easily express themselves verbally (Bell, 1996). Passy et al. (1993) found that patients began speaking almost immediately and their speech improved making it easier for them to communicate with hospital staff, doctors and family. This ease of communication is very beneficial to the patient's ability to direct their own care. Ability to participate easily and clearly are particularly important in decision making around interventions/management in dysphagia care.

The volume of secretions appears to increase when the PMV is removed (Passy et al., 1993; Lichtman et al., 1995). Manzano et al. (1993), found that patients experienced a decrease in secretions and showed improvement in ability to cough with the PMV in place. In studies conducted by Stachler et al. (1996) and Elpern et al. (2000), both noted that patients with the PMV showed significant improvement in degree of aspiration. Suiter et al. (2003) noted that individuals, once the valve was in place, were able to safely take thin liquids. Benefits of the PMV include: improved oxygenation, decreased oral and nasal secretions, improved sense of smell, enhanced airway clearance, and improved swallowing (Bell, 1996). To determine its effectiveness specifically within the ABI population more research is recommended.

5.10 Nutritional Management

5.10.1 Risk Factors for Malnutrition Post ABI

Q17. What are some unique risk factors for malnutrition in ABI patients?

Answers

1. Hypermetabolic state
2. Hypercatabolic state associated with additional injuries
3. Decreased level of consciousness

Discussion

The nutritional management of patients recovering from ABI presents many challenges. Despite the efforts of the clinicians, several factors make it difficult to avoid malnutrition in ABI patients, beginning with the metabolic changes that occur post-injury⁴³. In the event of ABI the damage to the metabolic control center causes more severe and protracted systematic responses than seen in many other forms of injuries, a possible consequence of the change in feedback mechanisms post-injury and the brain's critical role in triggering the metabolic response⁴⁴. Loan⁴⁵ noted that directly secondary to ABI, a catabolic and counterregulatory hormone (glucagons and cortisol) increase takes place. *"Deficiencies of follicle-stimulating hormones (FSH), leutenizing hormone (LH), and growth hormone (GH) indicate alteration in the hypothalamus-pituitary feed-back mechanism that normally regulates metabolism."*⁴⁵. Depending on the severity of the injury, nutritional requirements will be markedly increased while gastroparesis and ileus may delay the initiation of enteral nutritional support in mechanically ventilated patients.

As a result of hypermetabolism and hypercatabolism, both energy and protein requirements will be elevated in the first several weeks following injury. Negative energy and nitrogen balance, which may exceed 30 grams/day have been reported within the first week following injury⁴⁶⁻⁴⁹. Unfortunately, although muscle wasting occurs as a consequence of bed

rest and immobilization, only a portion of these losses are responsive to nutritional interventions⁵⁰.

Dénes⁵¹ stated that **rehabilitation problems associated with severely malnourished ABI patients include an increased occurrence of complications, a greater challenge in patient mobilization, an increased frequency for the need to operate on contractures and a longer length of stay in a rehabilitation unit.** Dénes⁵¹ concluded that there has been little research done regarding the complications and effects of malnutrition during rehabilitation.

5.7.2 The Incidence of Malnutrition

Q18. How common is malnutrition following ABI?

Answer

1. The incidence of obesity was comparable to normal.

Discussion

The incidence of malnutrition following ABI is difficult to estimate. No consistent criteria have been used to define it and relatively few studies have examined the issue. Given that accidental brain injuries tend to occur in younger, previously healthy individuals, it is unlikely that pre-existing nutritional deficits are prevalent at the time of injury. Therefore, declines in nutritional parameters are most likely directly related to the metabolic effects of the injury. However, substantial weight loss within the first several weeks has been reported and is certainly indicative of a compromised nutritional state. Brooke et al.⁵² reported an average weight loss of 13.2 kg from the time of injury to admission to a rehabilitation facility, while Weekes and Elia⁴⁷ also reported a weight loss from the time of injury to day 19 (9.8 kg) among 4 previously healthy young males. In the early rehabilitation phase, Brooke et al.⁵² reported that 60% of patients were considered underweight, while Haynes⁵³ reported 58%. However, obesity has also been reported among patients, typically in the chronic phase of recovery⁵⁴.

A single study was identified which reported the nutritional state of patients in the chronic phase of recovery⁵⁵. The mean time from injury to admission to the unit was almost 6 years. Among studies evaluating the nutritional status of patients in the acute phase of injury, only changes, which were typically declines, in nutritional parameters were reported. No studies attempted to classify patients as well or malnourished. Although they reported no malnutrition among a cohort of patients recovering from ABI, the authors did not define their criteria for malnutrition and used a body mass index (BMI) of 20 or greater to indicate the absence of nutritional deficit. Fifty-three percent of patients were classified as either overweight or obese and consumed more calories than required.

5.7.3 Hypermetabolism Post-ABI

Q19. Define Hypermetabolism Post ABI

Answer

1. An increase in metabolic rate above that predicted using equations, which take into account age, sex, height and weight.

2. Characterized by increased oxygen consumption and nitrogen excretion following injury.

Discussion

Hypermetabolism is a well known metabolic sequella of ABI. Hypermetabolism has been defined as an increase in metabolic rate above that which is predicted using equations, which take into account age, sex, height, and weight⁵⁶. The hypermetabolic state, which is characterized by increased oxygen consumption and nitrogen excretion following injury, is thought to be mediated by an increase in i) counterregulatory hormones such as epinephrine, norepinephrine and cortisol, ii) corticosteroids and iii) proinflammatory mediators and cytokines⁵⁷. Tremendous variability has been reported regarding the magnitude of the hypermetabolic state post ABI. The variations are likely due to the timing of the measurements, patient characteristics (initial level of injury, concomitant infections) and management (i.e. craniotomy, intubation and sedation and/or barbiturate use, ambient temperature).

5.8 Routes and Timing of Non-Oral Nutritional Interventions

5.8.1 Routes of Nutrient Administration

Q20. What would be the indications for enteral feeding? What would be the indications for parenteral feeding?

Answers

1. Enteral feeding is required when the patient is severely dysphagic, an aspirator, comatose or mechanically ventilated.
2. In these situations enteral feeding is the preferred option.
3. Parenteral feeding is indicated when enteral feeding is not possible – feeding intolerance due to gastroparesis and ileus as well as increased intracranial pressure.
4. Both parenteral and enteral feeding methods of nutrition administration safely and effectively reduce mortality and improve outcome following traumatic head injury; parenteral feeding was associated with .
5. There is conflicting evidence that choice of feeding route does not affect nitrogen balance or mortality in post-ABI patients.
6. Based on a single RCT, there is Level 2 evidence that TPN can safely be administered without causing serum hypersomolality or influencing intracranial (ICP) pressure levels or ICP therapy in post-ABI patients.

Discussion

In the early stages of recovery a significant percentage of patients will be comatose and mechanically ventilated, precluding oral feeding. While enteral feeding is the preferred route of nutrient administration, feeding intolerance due to gastroparesis and ileus are common. Enteral feeding has been associated with a decrease in bacterial translocation and a reduced incidence of infection.

Enteral feeding intolerance may be related to increases in intracranial pressure⁵⁸. Medications may also play a role in delayed gastric emptying. Although the placement of feeding tubes into the small bowel may theoretically improve tolerance, placement can be difficult and empirical

evidence of superiority is lacking. If intolerance is prolonged, parenteral feeding may be indicated⁵⁹ although the risk of hyperglycemia and cerebral edema are increased.

A Cochrane review authored by Yanagawa et al.⁶⁰ identified six trials, which **compared parenteral versus enteral nutrition. Parental feeding was associated with protection from both death and the combined outcome of death and disability**, although the result was not statistically significant. The relative risk for mortality at the end of the follow-up period was 0.66 (0.41-1.07) while two trials noted the relative risk of death and disability as 0.69 (0.40-1.15). Young et al.⁴⁴ noted that both parenteral and enteral feeding methods of nutrition administration safely and effectively reduce mortality and improve outcome following traumatic head injury.

Nutritional Recommendations (ABIKUS Guidelines 2007¹⁸)

All brain injured patients with significant ongoing impairment or disability should have their nutritional status assessed using a validated method, within 48 hours of admission (onset of injury). (ABIKUS B, adapted from RCP, G52, p.30) (G64-p.26)

Where patients are unable to maintain adequate nutrition orally, nutrition should be provided via nasogastric tube within 48 hours of injury, in collaboration with physician, dietician, nursing staff (ABIKUS A, adapted form RCP G53, p. 30) (G65-p.26)

A dietician trained in the management of brain injury should review nutrition and hydration needs regularly. This should include regularly weighing the patient (ABIKUS C, adapted from RCP, G55, p.30) (G66-p.26)

Nutritional needs may need to be changed according to changing metabolic demands (ABIKUS B, adapted from RCP, G54, p.30) (G67-p.26)

If the patient is unable to take adequate nutrition orally for longer than 2-3 weeks after injury, Percutaneous Endoscopic Gastrostomy (PEG) or similar intervention should be instituted, unless contradicted. (ABIKUS B, adapted from RCP, G56, p.30) (G68-p.26)

5.8.2 Enhanced Enteral Nutrition

Q21. What evidence is there of a benefit of enhanced enteral nutrition post ABI?

Answer

1. There is Level 1 evidence based on a single RCT that enhanced enteral nutrition enriched with immune-enhancing nutrients can reduce the incidence of infection, and reduce both the ventilator dependency period and ICU stay.

Discussion

Enteral feeding solutions enriched with immune-enhancing nutrients may decrease the occurrence of sepsis and reduce the inflammatory response. Theoretically, glutamine may improve the nutrition of both the gut mucosa and immune cells, while probiotic bacteria could

favourably alter the intraluminal environment, competing for nutrients and adhesion sites with pathogenic bacteria. These co-operative actions may reduce the rate of bacterial translocation and, thus, decrease both the incidence of infection and the length of hospitalization⁶¹.

5.8.3 Timing of Enteral Nutrition

Q22. What are the benefits of early administration of enteral nutrition post ABI?

Answers

1. There is Level 2 evidence based on a single RCT that initiating enteral feeding at goal rate will increase the percentage of prescribed energy and protein actually received.

Discussion

Early enteral feeding is desirable as a means to prevent intestinal mucosal atrophy and to preserve gut integrity, although, as previously noted, feeding intolerance occurs frequently. Three studies examined the effects of early vs. delayed enteral feeding. A Cochrane review authored by **Yanagawa et al.**⁶⁰ identified six RCTs, which addressed the timing to initiation of feeding and assessed mortality as an outcome. The relative risk for death associated with early nutritional support was 0.71 (95% CI 0.43-1.16). The pooled RR from three trials, which also assessed death and disability for early feeding was 0.75 (0.50-1.11). **Although the results were not statistically significant, the authors concluded that early feeding may be associated with a trend towards better outcomes in terms of survival and disability.**

5.8.4 Timing of Parenteral Nutrition

Q23. What are the benefits of early administration of parenteral nutrition post ABI?

Answer

1. There is Level 2 evidence that early parenteral nutrition support of closed head-injury patients appears to modify immunologic function by increasing CD4 cells, CD4-CD8 ratios, and T-lymphocyte responsiveness to Con A.

Discussion

Early parenteral nutrition support provided directly following injury could assist in the maintenance of immunocompetence and help reduce the frequency of the incidence of infection following acquired brain injury⁶². In a study conducted by Sacks et al.⁶² a significant increase in total CD4 cell counts and CD4% for early PN versus delayed PN at day 14 ($p < 0.05$) was found. From baseline to day 14, following Con A stimulation, an improved lymphocyte response was demonstrated in the early PN group ($p < 0.05$). The CD4-CD8 ratio significantly increased from baseline to day 12 in the early PN group ($p < 0.05$).

5.8.5 Types of Enteral Feeding Tubes

Q24. What evidence for one type of enteral feeding tube over another?

Answers

1. There is Level 1 evidence that the risk of developing pneumonia is higher among ventilated patients fed by a naso-gastric tube compared with a gastrostomy tube.
2. There is Level 2 evidence that early naso-jejunal hyperalimentation improves caloric intake, nitrogen intake, nitrogen balance, bacterial infection and days of stay in the intensive care unit in post-ABI patients.

Discussion

Early enteral feeding has been associated with improved outcome. However, the effectiveness of the intervention may vary depending on the mode of feeding. Nasogastric feeding tubes have been associated with increased incidence of pneumonia, while, theoretically feeding tubes placed more remotely decrease the risk. Gastronomies are proved to be a safe and dependable process used to provide enteral access for meeting nutritional needs of ABI patients and delivering essential medications ⁶³.

5.9 Miscellaneous Therapies

5.9.1 Zinc Supplementation

Q25. What evidence is there for zinc supplementation in ABI?

Answer

1. Based on a single RCT there is Level 1 evidence that zinc supplementation in ABI patients has a positive effect on neurological recovery as measured by the Glasgow Coma Scale. However, no significant improvement in mortality rates could be attributed to zinc supplementation.

Discussion

“Zinc is an essential element for humans that constitutes less than 0.1% of body weight, yet is vitally important for normal nucleic acid and protein metabolism” ⁶⁴. **Serum hypozincemia and increased urinary zinc excretion are common following head injury** and are thought to be an adaptive response to inhibit the proliferation of infective organisms. Levels of serum albumin, the major transport carrier for zinc, are also markedly depressed following brain injury and likely help to explain a portion of the reductions in serum zinc levels. Urinary excretion of zinc appears to be proportional to the severity of head injury ⁶⁵. Zinc is an important trace mineral in protein synthesis. Moderate zinc deficiency has been associated with cell death.

Two RCTs were identified which examined the effect of parenteral zinc supplementation following ABI ^{64,66}. Young et al., ⁶⁶ reported on improvements in protein synthesis and neurological recovery in patients who received supplementation. Surprisingly, there were no differences in either the serum or cerebrospinal fluid zinc concentrations between the groups.

5.9.2 Increased Nitrogen Feeds

Q26. What evidence is there of a benefit of enhanced enteral nutrition post ABI?

Answer

1. Based on a single RCT, there is Level 2 evidence that high nitrogen feedings of approximately 2 g protein/kg are necessary to restore the substantial nitrogen losses that occur post-ABI.

Discussion

Following brain injury, the incidence of metabolic changes will influence cell turnover, use of substrate and body composition⁶⁷. Twyman⁶⁷ noted that urinary urea nitrogen levels increase by a factor of three compared with normal levels within 10 days after severe head injury. On average, about 5 to 10 g of urea nitrogen are excreted daily from a normal individual; however, ABI patients' lose a mean of 21 g urinary urea in a single day⁶⁷. **Following brain injury, nitrogen losses result from the conversion of endogenous protein to energy with the extra stress demand**⁶⁸. Hadley et al.⁶⁹ also reported that attainment of a positive nitrogen balance is complicated because increasing the amount of nitrogen feeding will not be retained, rather it will cause an increased amount of nitrogen excretion. *"Positive nitrogen balance in brain injured patients usually does not occur until the catabolic stimulus begins to subside"*⁶⁹.

Reference List

- (1) Morgan AT, Ward EC. Swallowing: neuroanatomical and physiological framework. In: Murdoch BE, Theodoros DG, eds. *Traumatic brain injury: associated speech, language, and swallowing disorders*. San Diego: Singular Publishing Group; 2001;313-329.
- (2) Platt J. *Dysphagia management for long-term care: a manual for nurses and other healthcare professionals*. Hamilton, Ont: Clinical and educational services, 2001.
- (3) Field LH, Weiss CJ. Dysphagia with head injury. *Brain Inj* 1989;3:19-26.
- (4) Mackay LE, Morgan AS, Bernstein BA. Factors affecting oral feeding with severe traumatic brain injury. *J Head Trauma Rehabil* 1999;14:435-447.
- (5) Cherney LR, Halper AS. Swallowing problems in adults with traumatic brain injury. *Semin Neurol* 1996;16:349-353.
- (6) Ward EC, Morgan AT. Dysphagia following traumatic brain injury in adults and children: assessment and characteristics. In: Murdoch BE, Theodoros DG, eds. *Traumatic brain injury: associated speech, language, and swallowing disorders*. San Diego: Singular Publishing Group; 2001;331-367.
- (7) Terre R, Mearin F. Evolution of tracheal aspiration in severe traumatic brain injury-related oropharyngeal dysphagia: 1-year longitudinal follow-up study. *Neurogastroenterol Motil* 2009;21:361-369.
- (8) O'Neil-Pirozzi TM, Momose KJ, Mello J et al. Feasibility of swallowing interventions for tracheostomized individuals with severely disordered consciousness following traumatic brain injury. *Brain Inj* 2003;17:389-399.
- (9) Mackay LE, Morgan AS, Bernstein BA. Swallowing disorders in severe brain injury: risk factors affecting return to oral intake. *Arch Phys Med Rehabil* 1999;80:365-371.
- (10) Schurr MJ, Ebner KA, Maser AL, Sperling KB, Helgerson RB, Harms B. Formal swallowing evaluation and therapy after traumatic brain injury improves dysphagia outcomes. *J Trauma* 1999;46:817-821.
- (11) Linden P, Siebens AA. Dysphagia: predicting laryngeal penetration. *Arch Phys Med Rehabil* 1983;64:281-284.
- (12) Horner J, Massey EW. Silent aspiration following stroke. *Neurology* 1988;38:317-319.
- (13) Horner J, Massey EW, Riski JE, Lathrop DL, Chase KN. Aspiration following stroke: clinical correlates and outcome. *Neurology* 1988;38:1359-1362.
- (14) Splaingard ML, Hutchins B, Sulton LD, Chaudhuri G. Aspiration in rehabilitation patients: videofluoroscopy vs bedside clinical assessment. *Arch Phys Med Rehabil* 1988;69:637-640.
- (15) Muller-Lissner SA, Fimmel CJ, Will N, Muller-Duysing W, Heinzel F, Blum AL. Effect of gastric and transpyloric tubes on gastric emptying and duodenogastric reflux. *Gastroenterology* 1982;83:1276-1279.
- (16) Lazarus C, Logemann JA. Swallowing disorders in closed head trauma patients. *Arch Phys Med Rehabil* 1987;68:79-84.

- (17) Smithard DG, O'Neill PA, Parks C, Morris J. Complications and outcome after acute stroke. Does dysphagia matter? *Stroke* 1996;27:1200-1204.
- (18) Bayley M, Teasell R, Kua A, Marshall S, Cullen N, Colantonio A. *ABIKUS Evidence Based Recommendations for Rehabilitation of Moderate to Sever Acquired Brain Injury*. 1st ed. Ontario Neurotrauma Foundation, 2007.
- (19) Smith HA, Lee SH, O'Neill PA, Connolly MJ. The combination of bedside swallowing assessment and oxygen saturation monitoring of swallowing in acute stroke: a safe and humane screening tool. *Age Ageing* 2000;29:495-499.
- (20) Martino R, Pron G, Diamant N. Screening for oropharyngeal dysphagia in stroke: insufficient evidence for guidelines. *Dysphagia* 2000;15:19-30.
- (21) DePippo KL, Holas MA, Reding MJ. Validation of the 3-oz water swallow test for aspiration following stroke. *Arch Neurol* 1992;49:1259-1261.
- (22) Garon BR, Engle M, Ormiston C. Reliability of the 3 oz water swallow test utilizing cough reflex as sole indicator of aspiration. *J Neuro Rehab* 1995;9:143.
- (23) Chong MS, Lieu PK, Sitoh YY, Meng YY, Leow LP. Bedside clinical methods useful as screening test for aspiration in elderly patients with recent and previous strokes. *Ann Acad Med Singapore* 2003;32:790-794.
- (24) Lim SH, Lieu PK, Phua SY et al. Accuracy of bedside clinical methods compared with fiberoptic endoscopic examination of swallowing (FEES) in determining the risk of aspiration in acute stroke patients. *Dysphagia* 2001;16:1-6.
- (25) Wu MC, Chang YC, Wang TG, Lin LC. Evaluating swallowing dysfunction using a 100-ml water swallowing test. *Dysphagia* 2004;19:43-47.
- (26) Halper AS, Cherney LR, Cichowski K, Zhang M. Dysphagia after head trauma: the effect of cognitive-communicative impairments on functional outcomes. *J Head Trauma Rehabil* 1999;14:486-496.
- (27) Morgan AS, Mackay LE. Causes and complications associated with swallowing disorders in traumatic brain injury. *J Head Trauma Rehabil* 1999;14:454-461.
- (28) Tolep K, Getch CL, Criner GJ. Swallowing dysfunction in patients receiving prolonged mechanical ventilation. *Chest* 1996;109:167-172.
- (29) Passy-Muir Clinical Inservice Outline. *Passy-Muir Inc* [serial online] 2004.
- (30) Bach DB, Pouget S, Belle K et al. An integrated team approach to the management of patients with oropharyngeal dysphagia. *J Allied Health* 1989;18:459-468.
- (31) Milazzo LS, Bouchard J, Lund DA. The swallowing process: effect of aging and stroke. *Physical Medicine and Rehabilitation: state of the arts reviews* 1989;3:489-499.
- (32) Logemann JA. *Evaluation and treatment of swallowing disorders*. San Diego : College-Hill Press, 1983.
- (33) Donzelli J, Brady S, Wesling M, Craney M. Simultaneous modified Evans blue dye procedure and video nasal endoscopic evaluation of the swallow. *Laryngoscope* 2001;111:1746-1750.

- (34) Brady SL, Hildner CD, Hutchins BF. Simultaneous videofluoroscopic swallow study and modified Evans blue dye procedure: An evaluation of blue dye visualization in cases of known aspiration. *Dysphagia* 1999;14:146-149.
- (35) Belafsky PC, Blumenfeld L, LePage A, Nahrstedt K. The accuracy of the modified Evan's blue dye test in predicting aspiration. *Laryngoscope* 2003;113:1969-1972.
- (36) Heart and Stroke Foundation of Ontario. Improving recognition and management of dysphagia in acute stroke . 2002.
- Ref Type: Report
- (37) Langmore SE, Terpenning MS, Schork A et al. Predictors of aspiration pneumonia: how important is dysphagia? *Dysphagia* 1998;13:69-81.
- (38) Hoppers P, Holm SE. The role of fiberoptic endoscopy in dysphagia rehabilitation. *J Head Trauma Rehabil* 1999;14:475-485.
- (39) Winstein CJ. Neurogenic dysphagia. Frequency, progression, and outcome in adults following head injury. *Phys Ther* 1983;63:1992-1997.
- (40) Ward EC, Morgan AT. Rehabilitation of dysphagia following traumatic brain injury. In: Murdoch BE, Theodoros DG, eds. *Traumatic brain injury: associated speech, language, and swallowing disorders*. San Diego: Singular Publishing Group; 2001;369-401.
- (41) Logemann JA. Behavioral management for oropharyngeal dysphagia. *Folia-Phoniatica-et-Logopedica* 1999;51:199-212.
- (42) Logemann JA. Approaches to management of disordered swallowing. *Bailliere's Clinical Gastroenterology* 1991;5:269-280.
- (43) Elovic E. Pharmacological therapeutics in nutritional management. *J Head Trauma Rehabil* 2000;15:962-964.
- (44) Young B, Ott L, Yingling B, McClain C. Nutrition and brain injury. *J Neurotrauma* 1992;9 Suppl 1:S375-S383.
- (45) Loan T. Metabolic/nutritional alterations of traumatic brain injury. *Nutrition* 1999;15:809-812.
- (46) Young B, Ott L, Norton J et al. Metabolic and nutritional sequelae in the non-steroid treated head injury patient. *Neurosurgery* 1985;17:784-791.
- (47) Weekes E, Elia M. Observations on the patterns of 24-hour energy expenditure changes in body composition and gastric emptying in head-injured patients receiving nasogastric tube feeding. *JPEN J Parenter Enteral Nutr* 1996;20:31-37.
- (48) Bruder N, Lassegue D, Pelissier D, Graziani N, Francois G. Energy expenditure and withdrawal of sedation in severe head-injured patients. *Crit Care Med* 1994;22:1114-1119.
- (49) Wilson RF, Dente C, Tyburski JG. The nutritional management of patients with head injuries. *Neurol Res* 2001;23:121-128.
- (50) Behrman SW, Kudsk KA, Brown RO, Vehe KL, Wojtysiak SL. The effect of growth hormone on nutritional markers in enterally fed immobilized trauma patients. *JPEN J Parenter Enteral Nutr* 1995;19:41-46.

- (51) Denes Z. The influence of severe malnutrition on rehabilitation in patients with severe head injury. *Disabil Rehabil* 2004;26:1163-1165.
- (52) Brooke MM, Barbour PG, Cording LG et al. Nutritional status during rehabilitation after head injury. *J Neurol Rehab* 1989;3:27-33.
- (53) Haynes MKM. Nutrition in the severely head-injured patient. *Clin Rehab* 1992;6:153-158.
- (54) Henson MB, De Castro JM, Stringer AY, Johnson C. Food intake by brain-injured humans who are in the chronic phase of recovery. *Brain Inj* 1993;7:169-178.
- (55) French AM, Merriman SH. Nutritional status of a brain-injured population in a long-stay rehabilitation unit: a pilot study. *J Hum Nutr Diet* 1999;12:35-42.
- (56) Souba WW, Wilmore DW. Diet and nutrition in the care of the patient with surgery, trauma, and sepsis. In: Shils ME, Olson JA, Shike M, Ross AC, eds. *Modern nutrition in health and disease*. 9th ed. Baltimore, MD: Williams and Wilkins; 1999;1589-1618.
- (57) Pepe JL, Barba CA. The metabolic response to acute traumatic brain injury and implications for nutritional support. *J Head Trauma Rehabil* 1999;14:462-474.
- (58) Ott L, Young B, Phillips R, McClain C. Brain injury and nutrition. *Nutr Clin Pract* 1990;5:68-73.
- (59) Cerra FB, Benitez MR, Blackburn GL et al. Applied nutrition in ICU patients. A consensus statement of the American College of Chest Physicians. *Chest* 1997;111:769-778.
- (60) Yanagawa T, Bunn F, Roberts I, Wentz R, Pierro A. Nutritional support for head-injured patients. *Cochrane Database Syst Rev* 2002;CD001530.
- (61) Falcao dA, I, Aguiar-Nascimento JE. Benefits of early enteral nutrition with glutamine and probiotics in brain injury patients. *Clin Sci (Lond)* 2004;106:287-292.
- (62) Sacks GS, Brown RO, Teague D, Dickerson RN, Tolley EA, Kudsk KA. Early nutrition support modifies immune function in patients sustaining severe head injury. *JPEN J Parenter Enteral Nutr* 1995;19:387-392.
- (63) Harbrecht BG, Moraca RJ, Saul M, Courcoulas AP. Percutaneous endoscopic gastrostomy reduces total hospital costs in head-injured patients. *Am J Surg* 1998;176:311-314.
- (64) McClain CJ, Twyman DL, Ott LG et al. Serum and urine zinc response in head-injured patients. *J Neurosurg* 1986;64:224-230.
- (65) Levenson CW. Zinc supplementation: neuroprotective or neurotoxic? *Nutr Rev* 2005;63:122-125.
- (66) Young B, Ott L, Kasarskis E et al. Zinc supplementation is associated with improved neurologic recovery rate and visceral protein levels of patients with severe closed head injury. *J Neurotrauma* 1996;13:25-34.
- (67) Twyman D. Nutritional management of the critically ill neurologic patient. *Crit Care Clin* 1997;13:39-49.
- (68) Graham TW, Zadrozny DB, Harrington T. The benefits of early jejunal hyperalimentation in the head-injured patient. *Neurosurgery* 1989;25:729-735.

- (69) Hadley MN, Grahm TW, Harrington T, Schiller WR, McDermott MK, Posillico DB. Nutritional support and neurotrauma: a critical review of early nutrition in forty-five acute head injury patients. *Neurosurgery* 1986;19:367-373.