

E. Medical Complications

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E1. Dysphagia

E1.1 Introduction to Dysphagia Post Stroke

Q1. Define dysphagia.

Answer

1. Dysphagia is defined as difficulty with swallowing

Q2. Why is dysphagia important following a stroke?

Answer

1. Dysphagia is common following a stroke.
2. Incident rates are high following an acute stroke
3. Associated with increased mortality and morbidity such as malnutrition, dehydration and pneumonia.
4. Diagnosing and treating dysphagia in stroke patient improves outcomes such as reduced risk of pneumonia, length of hospital stay and overall health expenditures.
5. Aspiration, the most important clinical consequence of dysphagia, has long been associated with an increased risk of pneumonia, death and sepsis.

E1.2 The Normal Swallowing Process

Q3. Describe the normal swallowing process.

Answers

Normal swallowing consists of 4 phases:

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

E1.3 Defining Dysphagia and Aspiration

Q4. What is the difference between Dysphagia and Aspiration?

Answers

1. Dysphagia is defined as difficulty with swallowing.

2. Aspiration is defined as entry of oral or gastric material into the airway below the level of the true vocal cords.

E1.4 Risk Factors for Aspiration Post Stroke

Q5. What are the risk factors or clinical red flags for aspiration post stroke?

Answer

1. Brainstem Stroke
2. Difficulty swallowing oral secretions
3. Coughing/throat clearing or wet, gurgly voice quality after swallowing water
4. Choking more than once while drinking 50 cc of water
5. Weak voice and cough
6. Wet-hoarse voice quality
7. Recurrent lower respiratory infections
8. Low-grade fever or leukocytosis
9. Auscultatory evidence of lower lobe congestion
10. Immunocompromised state

E1.5 Dysphagia Post Stroke

Q6. How common is dysphagia following acute stroke?

Answer

1. The incidence of dysphagia appears to be quite high following acute stroke with between one third and two-thirds of patients affected, depending on the population studied and the criteria used to diagnose dysphagia.

E1.6 Silent Aspiration

Q7. What is silent aspiration and why is it important?

Answers

1. Silent aspiration is defined as “penetration of food below the level of the true vocal cords without cough or any outward signs of difficulty”.
2. Important because aspiration risk may go undetected placing the patient at higher risk of developing aspiration related complications.
3. 9-27% of acute stroke patients are silent aspirators, a condition which is only reliably detectable through VMBS studies.
4. Between one-third to one-half of aspirators are “silent” aspirators.

Q8. When should silent aspiration in a stroke patient be suspected?

Answer

1. Silent aspiration should be suspected in any stroke patient who presents with recurrent lower respiratory infections, chronic congestion, a low-grade fever, leukocytosis, weak voice or cough or a wet-hoarse voice quality after swallowing.

Canadian Stroke Strategy Guideline (2008): Recommendation 6.1 – Dysphagia Assessment

Patients with stroke should have their swallowing ability screened using a simple, valid, reliable bedside testing protocol as part of their initial assessment, and before initiating oral intake of medications, fluids or food [Evidence Level B] (CSQCS, NZ, SCORE, SIGN 78).

- i. Patients who are not alert within the first 24 hours should be monitored closely and dysphagia screening performed when clinically appropriate [Evidence Level C].
- ii. Patients with stroke presenting with features indicating dysphagia or pulmonary aspiration should receive a full clinical assessment of their swallowing ability by a speech–language pathologist or appropriately trained specialist who should advise on safety of swallowing ability and consistency of diet and fluids [Evidence Level A] (CSQCS, NZ, RCP, SCORE).
- iii. Patients who are at risk of malnutrition, including those with dysphagia, should be referred to a dietitian for assessment and ongoing management. Assessment of nutritional status should include the use of validated nutrition assessment tools or measures [Evidence Level C] (AU).

E1.7 Pneumonia and Aspiration Post Stroke

Q9. What is the relationship between aspiration and pneumonia?

Answers

1. Aspiration alone is not sufficient to cause pneumonia. Aspiration of small amounts of saliva occurs during sleep in almost half of normal subjects.
2. Aspiration pneumonia is thought to occur when the lung's natural defences are overwhelmed when excessive and/or toxic gastric contents are aspirated, leading to a localized infection or a chemical pneumonitis.
3. Those patients who aspirate over 10% of the test bolus or who have severe oral and/or pharyngeal motility problems on VMBS studies are considered at high risk for pneumonia.
4. The severity of aspiration correlates with the risk of developing pneumonia.

E1.8 Risk Factors for Aspiration Pneumonia

Q10. What are some of the risk factors for aspiration pneumonia?

Answers

1. Brainstem stroke
2. Aspiration of VMBS (risk greater if greater than 10% aspirated)
3. Aspiration of thick fluids or solids
4. Slower pharyngeal transit time on VMBS
5. Reduced levels of consciousness
6. Elderly
7. Dysarthria
8. Cognitive difficulties
9. Failure of water swallow test

E2. Dysphagia Case Study

E2.1 Lateral Medullary Infarction (Wallenburg's Syndrome)

Case Study

A 68-year old man presents with a stroke involving the territory of the left lateral medulla. This is due to an infarct of the posterior inferior cerebellar artery. The patient presents to the Emergency Room with significant ataxia, dizziness and dysarthria.

Q1. Describe the affected vasculature and the typical presentation of a left lateral medullary infarction (Wallenburg's syndrome)?

Answers

Ipsilateral Side (right-side):

- Horner's syndrome (ptosis, anhidrosis and miosis)
- Decrease in pain and temperature over ipsilateral face
- Cerebellar signs such as ataxia

Contralateral Side (left-side):

- Decreased pain and temperature over contralateral body
- Dysphagia, dysarthria, hoarseness and paralysis of vocal cord
- Vertigo, nausea and vomiting
- Hiccups
- Nystagmus, diplopia

Should not mention facial or extremity weakness.

E2.2 Assessment of Dysphagia Post Stroke

Case Study (continued)

The nurse in the emergency room provides the patient with a drink of water. The patient responds with choking. He develops a persistent cough while in the emergency room.

Q2. What would be the next step?

Answers

1. Having had a brainstem stroke, this patient would be considered at relatively high risk of aspirating.
2. Acute stroke patients should be maintained NPO until their swallowing ability is determined.
3. A clinical bedside screening should be conducted by a trained team member and if there are any concerns or the patient is regarded at risk of dysphagia than an oral motor assessment should be performed, generally by a speech-language pathologist.
4. This trial involves 1-2 teaspoons of water and if that is well tolerated is followed by a small cup of water.

Case Study (continued)

The nurse looking after the patient on the acute floor has concerns that when given some water the patient again chokes. The patient manages the 2 teaspoons of water without difficulty but chokes once on a small cup of water (50ml) and his voice now has a wet, gurgly sound to it.

Q3. Discuss management options now.

Answers

1. Once a patient fails a screening test and it has been determined that a problem exists, a more comprehensive assessment should follow, from which treatment options are determined.
2. The assumption needs to be made that the patient is an aspirator and a videoscopic modified barium swallow (VMBS) needs to be performed.
3. While the patient is waiting for the VMBS, the patient is kept NPO and an nasogastric (NG) tube is inserted to ensure that the patient receives adequate nutrition and hydration.

E2.3 Management of Dysphagia Post Stroke

Case Study (continued)

The patient aspirates all consistencies of barium laced food on VMBS but only coughs on the thin liquids. The video modified barium swallow shows significant aspiration due to poor coordination of the pharyngeal phase of swallowing (>10% of the test bolus).

Q4. Discuss management strategies.

Answers

1. The patient should not be fed by mouth and an NG tube is required for an extended period of time.
2. At this point many centers would install a GJ tube to facilitate maintenance of nutrition and hydration rather than wait.

Case Study (continued)

The patient has a GJ tube installed. A second modified barium swallow is done and this shows an improvement in the aspiration (<10% of test bolus). The patient is able to handle pureed consistencies well but still shows aspiration on thin liquids.

Q5. Discuss management.**Answers**

1. The patient can be fed orally a pureed diet with a restriction on thin liquids.
2. Fluids can be supplemented through the G-J tube.
3. Another VMBS would need to be ordered for 2-3 months and the patient carefully monitored by a dietician and speech-language pathologist.

Case Study (continued)

3 months later the patient has a repeat VMBS and continues to show improvement, although she is still having trace aspiration with thin liquids.

Q6. Discuss management.**Answers**

1. The patient can continue to be fed orally an unrestricted diet.
2. However, they are advised to sip thin liquids and double cough afterwards.
3. The patient needs to continue to be monitored for a lung infection.

E3. Dysphagia Case Study

Case Study

A 56-year-old right-handed hypertensive, type II diabetic was admitted to a local hospital with complaints of occipital headache, right hand tingling, right limb ataxia, hoarse voice, dysphagia, nausea, vomiting and vertigo. Blood pressure was elevated but weakness was not noted on examination. He was transferred to a tertiary care center where an occlusion of the posterior inferior cerebellar artery was diagnosed.

E3.1 Lateral Medullary Infarction (Wallenburg's syndrome)

Q1. Describe the affected vasculature and the typical presentation of a right lateral medullary infarction (Wallenburg's syndrome)?

Ipsilateral Side (right-side):

- Horner's syndrome (ptosis, anhydrosis and miosis)
- Decrease in pain and temperature over ipsilateral face
- Cerebellar signs such as ataxia

Contralateral Side (left-side):

- Decreased pain and temperature over contralateral body
- Dysphagia, dysarthria, hoarseness and paralysis of vocal cord
- Vertigo, nausea and vomiting
- Hiccups
- Nystagmus, diplopia

Should not mention facial or extremity weakness.

E3.2 Management of Dysphagia Post Stroke

Case Study (continued)

The following day he was noted to have severe dysphagia which was his primary complaint and bilateral aspiration pneumonia which was confirmed on x-ray. Antibiotics were initiated for the pneumonia. The speech language pathologist did a bedside swallowing assessment on the day of admission. His vocal quality was described as "wet" and he could not elevate the hyoid bone, indicating a probable swallow reflex problem. Oral motor elevation revealed tongue elevation was significantly reduced, with a low resting soft palate on the left and reduced movement on that side during phonation. No gag reflex

could be elicited. Mild hypernasality during conversation was noted. Maximum phonation duration at 6 seconds indicated reduced breath support, likely resulting from vocal cord paralysis. During phonation and conversation vocal quality was moderately breathy. He was noted to have a weak cough and diminished throat-clearing ability. A swallow reflex could not be elicited.

Q2. What step would you do next?

Answers

1. The next step would be to decide to do a VMBS or if not readily available, assume the patient was a gross aspirator.

Case Study (continued)

The bedside swallow assessment therefore deemed he was unsafe for oral feeds and at risk for the development of further aspiration. A VMBS was not performed at that time because it was not perceived that the test would change clinical management, in view of the severity of the results of the bedside assessment.

Initially the patient was kept NPO and a NG tube was inserted to provide nonoral feeds. The patient was carefully followed and one week after the stroke the SLP noted evidence of initiation of a swallow reflex. At that point, a clinical trial of both thin and thick fluids was attempted. He continued to demonstrate clinical signs of possible aspiration, such as coughing and a wet voice with both fluid consistencies. There was a delay in the initiation of the swallow reflex and the patient reported marked difficulty in clearing food through the pharynx. Vocal quality was wet and gurgly and there was post-swallowing coughing and throat clearing. A head turn compensatory strategy toward the left side was attempted to direct the bolus down the stronger side of the pharynx. The patient reported that this seemed to only minimally help the pharyngeal clearing. Given the severity of his swallowing problems, the apparent high risk of aspiration, and the anatomical location of his stroke, a GJ tube was recommended and this was inserted percutaneously.

During the first month following his stroke, the patient demonstrated improvement in his pharyngeal swallow including better clearing of oral secretions and mild improvement in clearing of thickened fluids. He continued to use lateral head rotation to the left side during the swallow assessment; however, there were continuing clinical signs of laryngeal penetration of oral contents.

The patient was subsequently admitted to a stroke rehabilitation unit one month following the onset of his stroke. Further bedside assessments indicated that he was unable to tolerate more than half a teaspoon of thin liquids at any time. The SLP noted that the patient likely had experienced a delay in the initiation of his pharyngeal swallow, and he was experiencing weakness of pharyngeal peristalsis. This likely resulted in residue in the both valleculae and pyriform sinuses. The patient was given oral and pharyngeal exercises to be used when clearing his oral secretions to improve the strength of pharyngeal constriction, i.e., hard glottal swallows with the head turned to the left.

Q3. What would be your next step?

Answer

1. Next step would be to conduct a repeat VMBS study (see the algorithm below).

Case Study (continued)

A VMBS study was subsequently performed and indicated good oral transport of all consistencies presented. A half teaspoon of pudding revealed moderate delay in the swallow reflex with minimal residue in the valleculae but a large residue present in the pyriform sinus that was then grossly aspirated. Presentation of a half teaspoon of thick liquids with the chin tucked again revealed gross aspiration occurring from a large residue in the pyriform sinus. A second half teaspoon of thick liquids with head turned to the left continued to show large residual in the pyriform sinus along with gross aspiration. It was recommended that the patient continue with the GJ tube feedings exclusively. He was receiving tube feedings from 1900 to 0800 hours and at 1200 to 1330 hours. This schedule was designed to minimize disruption to rehabilitation therapies.

At the time of discharge from the stroke rehabilitation program almost 2 months after his initial presentation with the stroke, the patient was close to be an independent with a walker, requiring only minimal assistance with tub transfers and being discharged home with the G-J tube feedings.

Q4. What would be the next step?

Answers

1. The next step would be to carefully follow the patient and later repeat the VMBS hoping to get this gentleman off of the G-J tube feedings.

Case Study (continued)

A second VMBS was performed 4 months post-stroke onset. This showed that the pudding and thick fluids still resulted in large residue being present in the pyriform sinus with moderate to large aspiration from both. No cough was heard when he aspirated. With thin liquids large residue was present in the pyriform sinus resulting in laryngeal penetration and aspiration. This VMBS study indicated that he was still unsafe with swallowing and required tube feedings. Upon laryngeal penetration of thick liquids he was asked to cough and this cleared the penetration. A third VMBS was conducted 2 months later (6 months post-stroke onset). Poor epiglottic motion and weak pharyngeal peristalsis were present. It was noted that there was marked pyriform sinus residue and mild vallecular residue with thin and thick liquid barium and pudding consistencies. Aspiration was demonstrated with these consistencies but they did not elicit a cough. It was recommended that he continue to be fed through the GJ tube.

Six months later (1 year subsequent to his lateral medullary stroke) this man had his fourth VMBS study. The SLP noted a significant improvement in his swallowing. He continued to show large residue in the valleculae and pyriform sinuses, more so in the latter. Double swallowing with consistencies of thick liquids and pudding allowed him to clear the residue. He was able to swallow small amounts of thin liquids, with double swallowing, with no aspiration. He had trouble with bread and cookie consistencies.

Q5. How would you manage his swallowing problem at this stage?

Answers

1. The patient should be placed on a pureed diet with thick fluids but not allowed bread or cookie consistencies
2. Care needs to be taken with thin liquids and it would be best to not allow him to drink thin liquids but continue to provide additional fluid through the G-J tube.

Case Study (continued)

The patient was put on a pureed diet with thick fluids but was not allowed bread or cookie consistencies. He was still maintained on GJ tube feedings primarily at need, but this was reduced to allow for his oral feedings.

A fifth and final VMBS was performed 6 months later, more than 18 months following the onset of his stroke. Most of the thick liquid barium was transferred into the esophagus; however, there was impaired pharyngeal peristalsis so that after swallowing a minimal residue remained in the valleculae and a minimum to moderate residue remained in the pyriform sinus. No laryngeal penetration was noted. With pudding, bread, and cookie consistencies the results were the same. With thin liquids there was occasional episodes of trace laryngeal penetration and trace aspiration. After the swallows minimal residue was in the valleculae and minimum to moderate residue was in the pyriform sinus. Results with thin liquids from a cup were similar.

Q6. What would you do at this stage?

Answers

1. The patient can now be fed orally for all consistencies.
2. Care still needs to be taken with thin liquids with the patient double swallowing and throat clearing after the fact.
3. He would need to be carefully monitored for any future episodes of pneumonia.

Case Study (continued)

It was noted that despite abnormalities in the pharyngeal phase of swallowing, there was no laryngeal penetration or aspiration with thick fluids, pudding, bread or cookie consistencies. It was suggested that the patient continue to use supraglottic swallowing as well as throat clearing to remove any material that may have penetrated. It was recommended that he be placed on a diet of thin liquids and regular solids and no further interventions were felt to be necessary. The GJ tube was subsequently removed. At one time during his post-stroke period, cloxacillin was ordered for a subcutaneous skin infection around the GJ tube.

E4. Dysphagia Management Post Stroke in Nursing Home Patient

E4.1 Low-Risk Feeding Strategies

Case Study

You are asked to see an 82 year old female who had a large right hemispheric stroke 2 years previously and who is now in a nursing home. Initially, while in hospital she had trouble with dysphagia, initially had an NG tube in place but eventually graduated to a regular diet. She has a left spastic hemiparesis, can ambulate with a cane and the assist of one person, does not have use of her left arm and hand, and still tends to neglect items on the left side. She has recently suffered two episodes of pneumonia, both of which necessitated admission to an acute care hospital.

You are able to observe her eating lunch on the day you arrive at the nursing home. Lunch is served in a large room with a number of the nursing home residents which is regarded as important to ensuring residents are able to regularly socialize. The patient herself is a very slow eater and so the nursing home has kindly assigned young students to help feed her. Initially, the patient was able to feed herself but increasingly she has come to depend largely on help with feeding. She is provided with a regular diet. She sits in her chair, frequently leaning back and watching patients and staff, mostly on her right side. The students are attentive and knowing that she tires easily, they try to get her to eat as much as possible before she asks to go back to her room. Hence, they feed her utilizing a tablespoon until she complains of being tired at which point she is taken to her room and allowed to lie down and rest. Her family was pleased at the attention given her to ensure she receives adequate nutrition and a couple of times per week they will assist.

Q1. What recommendations would you make with regard to feeding strategies?

Answers

1. The patient should be in a quiet environment.
2. The patient should be sitting at a 90 degree angle with the neck in a slightly flexed forward position.
3. The patient should be fed by seated individual at eye level with the patient.
4. The patient should be fed slowly with a teaspoon – no tablespoon.
5. The feeding assistant should assure that the patient swallows oral contents before offering more food.
6. Once done the patient should be kept upright for a half-hour.

E5. Nutritional Issues Following Stroke

Case Study

A 75-year old female suffered a small subcortical stroke resulting in dysarthria and clumsy hand syndrome. On admission to rehabilitation 10 days following the onset of symptoms, the patient was observed to appear thin and had only been consuming about half of her meal trays while on the acute service. She was safe with a regular diet. A dietitian was consulted and an assessment was completed within several days.

History: She has been living on her own for several years in a seniors apartment since her husband died. She rarely cooks anymore but receives 4 hours a week of home-care services and has meals provided by Wheels (an external agency) 3x/week. She does not have a scale and is unsure if she has lost any weight over the past 6 months. She ambulates independently but uses a category II walker for safety. She reports her appetite is "fair". She has no problems with nausea or vomiting.

Physical: Ht: 160 cm wt: 47 kg. (6'3", 103.4 lbs.) Her shoulders have a squared-off appearance. There is loss of fat in the interosseous and palmar areas of the hand.

Biochemistry: Normal Reference range included in parentheses

Total protein - 70 g/L (60-80 g/L)

Serum albumin - 37 g/L (>35 g/L)

Serum prealbumin - 0.20 g/L (>0.18 g/L)

Serum creatinine - 4 g/L (8 to 14 g/L)

Random glucose - 6 mmol/L (<11 mmol/L)

E5.1 Body Mass Index (BMI)

Q1. What is this patient's Body Mass Index (BMI)?

Answers

BMI or body mass index is often used to estimate whether a person is underweight, normal weight or overweight. While there are limitations associated with the use of BMI to detect overweight or obese individuals, it is useful to help quickly identify those who are underweight and may be malnourished.

$$\text{BMI} = \text{Weight [in kilograms]} / (\text{Height [in meters]})^2$$

$$\text{BMI} = 47 / 1.60^2$$

BMI = 18.4

There are many interpretations of BMI although values between 19.5 and 23.5 are considered optimum by most professionals. A BMI of 25 to 29.9 is considered overweight and one 30 or above is considered obese. A value less than 19.5 is considered to be underweight.

E5.2 Assessment of Nutritional State

Q2. What information can be used from her history and physical to help with the assessment of her nutritional state?

Answers

1. Based on the limited information given, we can assume that both her remote and recent intake have been less than optimum. She has been living on her own and doesn't cook for herself regularly. Her oral intake while in the hospital appears to be poor. Her low BMI also supports this finding.
2. Her physical appearance suggests evidence of both muscle wasting and fat loss.
3. The *biochemical data* we are given reveals few abnormalities; other than a low serum creatinine value.

Q3. What is this woman's nutritional status?

1. This woman presents with classic protein-energy malnutrition (PEM), or marasmus.
2. Key indicators are a low BMI, history of poor intake and a wasted appearance. It is important to note that in this condition, there are few abnormal biochemical values. Visceral proteins, such as albumin, frequently used to assess nutritional status, are often spared at the expense of skeletal muscle, which is used to help meet energy requirements. The low serum creatinine (4 g/L) suggests a loss of muscle mass, consistent with a low BMI.

Q4. Name some other nutrition assessment tools that can be used?

1. Subjective Global Assessment.
2. Mini-Nutritional Assessment.

Q4-1. Describe the Subjective Global Assessment.

Answer

The SGA is a method of nutritional assessment that was designed for use in the prediction of risk for complications following general surgery, based on pre-operative nutritional state (Detsky et al. 1987).

Q4-2. Describe the Mini-Nutritional Assessment.

Answer

The MNA was developed as a screening and assessment tool to identify geriatric patients at risk for malnutrition (Guigoz et al. 1994).

E5.3 Protein and Energy Requirements

Q5. What are this patient's protein and energy requirements?

Answers

There are a number of different methods to estimate energy requirements. One of the most commonly used is the Harris Benedict equation, which estimates resting energy requirements using a patient's height, weight and age

$$\begin{aligned} \text{M: } & 66.5 + 13.8(W) + 5(H) - 6.8(\text{age}) = \text{Kcal/day} \\ \text{F: } & 655 + 9.6(W) + 1.8(H) - 4.7(\text{age}) = \text{Kcals/day} \end{aligned}$$

$$\begin{aligned} \text{This patient would require } & 1,047 \text{ Kcals/day} \\ & 655 + 9.6(103) + 1.8(63) - 4.7(75) \end{aligned}$$

A stress factor of 1.2 combined with an activity factor of 1.2 are used to estimate the total amount of energy required for repletion

$$1,047 \times 1.2 \times 1.2 = 1,508 \text{ Kcals/day}$$

A simpler method of estimating energy intake is to use a standard requirement of 35 Kcals/kg

$$35 \text{ Kcals/day} \times 47 \text{ kg} = 1,645 \text{ Kcals/day to achieve weight gain}$$

Protein requirements are estimated to be 1-1.2 g/kg/day (47-56 g/day).

Q6. What diet would you recommend for this patient? What are the goals of treatment?

Answers

1. Given that this woman appears to be suffering from uncomplicated malnutrition and does not require dietary restrictions due to dysphagia or co-morbidities, a *high calorie, high protein diet* is indicated.

2. Weight gain is the goal of treatment.

Q7. How do you monitor and assess the progress?

Answers

1. *Weekly weights* should be obtained for the duration of the inpatient stay.
2. *Calorie counts* should be continued intermittently to ensure that adequacy of intake is maintained.

Reference

Detsky AS, McLaughlin JR, Baker JP, et al. What is subjective global assessment of nutritional status? JPEN 1987; 11:8-13

Guigoz Y, Vellas B, Garry P. The Mini Nutritional Assessment: A practical assessment tool for grading the nutritional state of elderly patients. Facts Res Gerontol 1994; 4:15-59.

E6. Deep Venous Thromboembolism

Canadian Stroke Strategy Guidelines 2008: Recommendation 4.2a – Venous Thromboembolism Prophylaxis

All stroke patients should be assessed for their risk of developing venous thromboembolism (including deep vein thrombosis and pulmonary embolism).

Patients considered as high risk include patients with inability to move one or both lower limbs and those patients unable to mobilize independently.

- i. Patients who are identified as high risk for venous thromboembolism should be considered for prophylaxis provided there are no contraindications [Evidence Level B] (ESO).
- ii. Early mobilization and adequate hydration should be encouraged with all acute stroke patients to help prevent venous thromboembolism [Evidence Level C] (AU, ESO, SCORE).
- iii. The use of secondary stroke prevention measures, such as antiplatelet therapy, should be optimized in all stroke patients [Evidence Level A] (ASA, AU, NZ, RCP, SIGN 13).
- iv. The following interventions may be used for patients with acute ischemic stroke at high risk of venous thromboembolism in the absence of contraindications:
 - a. low molecular weight heparin (with appropriate prophylactic doses per agent) or heparin in prophylactic doses (5000 units twice a day) [Evidence Level A] (ASA, AU, ESO);
 - b. external compression stockings [Evidence Level B] (AU, ESO).
- v. For patients with hemorrhagic stroke, nonpharmacologic means of prophylaxis (as described above) should be considered to reduce the risk of venous thromboembolism [Evidence Level C].

Case Study

A 75 year-old man presents to the Emergency-Room with left hemiplegia, due to a right MCA stroke. 48 hours later the nurse alerts you to the fact that his left leg shows signs of swelling and erythema.

Q1. What do this patient's symptoms suggest?

Answer

1. This patient is showing signs of deep venous thrombosis (DVT).
2. The clinical features of DVT are present in less than half of patients with this condition.

E6.1 Incidence of Venous Thromboembolism in Stroke

Q2. Describe the incidence and impact of DVT in the stroke population in the absence of prophylaxis.

Answers

1. In the absence of prophylaxis, over 60% of dense hemiplegics develop DVTs, 9-15% suffer a pulmonary embolus, with a 1-2% mortality rate (Sioson et al. 1988).
2. In the acute phase, in the absence of prophylaxis the incidence is 50%.
3. Most are below the knee, asymptomatic and unlikely to lead to a pulmonary embolus.
4. Peak onset is 2 to 7 days post stroke.
5. The incidence of DVT on rehabilitation is less than 10% in the rehabilitation phase in the absence of prophylaxis.

E6.2 Risk Factors for Venous Thromboembolism Post Stroke

Q3. The nurse asks you how one can tell which patients are at risk for developing a DVT?

Answers

1. Risk factors include degree of lower limb paralysis, older age, reduced consciousness, obesity, previous DVT and atrial fibrillation.

E6.3 Diagnosis of Deep Venous Thrombosis

Q4. What diagnostic tests would you initially use to confirm a clinical suspicion of a DVT? Describe briefly the role of each.

Answers

1. D-Dimer Assay: D-dimers are fibrin degradation products. Very sensitive but lacks specificity.
2. Venous Ultrasound: Test is noninvasive and can do serial testing; sensitivity 95% for proximal DVTs and 73% for distal DVTs.

Q5. How can you make a definitive diagnosis of a DVT?

Answer

1. Venography
2. MRI

E6.4 Prevention of Deep Venous Thrombosis Post Stroke

Q6. What is the evidence for preventive pharmacological treatments for DVT recommended in ischemic strokes?

Answers

1. There is strong evidence that prophylactic anticoagulation significantly reduces the incidence of deep venous thromboembolism, as compared to placebo.
2. Although there is a slightly higher risk of intracerebral hemorrhage, the benefit of prophylaxis far outweighs the risk.

Q7. The nurse wants to know which pharmacological agents are recommended for DVT prophylaxis?

Answer

1. There is strong evidence that low molecular weight heparin is more effective with less risk of hemorrhagic complications than unfractionated heparin.
2. Warfarin is an effective anticoagulant but is less reliable, more cumbersome to use and has more bleeding complications than LMW heparin for prophylaxis.

Q8. A resident asks you about the use of mechanical methods for preventing DVT, what can you tell them?

Answers

1. There are two physical forms of prophylaxis for DVT: graded compression stockings and intermittent pneumatic calf compression devices.
2. For both types, there is a moderate level of evidence that they are ineffective at reducing the risk of developing DVT.

E6.5 Pulmonary Embolism

Case Study (continued)

You receive another call from the nurse who tells you that the patient is now experiencing chest pain, shortness of breath, and is not interested in participating in rehabilitation therapies.

Q9. What is the most likely diagnosis and how common is it in the stroke population?

Answer

1. Most likely a pulmonary thromboembolism
2. Relatively common following a stroke with leg paresis
3. Most severe and fatal PEs occur at 2-4 weeks post stroke.

Q10. The nurse asks you how she can tell which patients are at risk for developing a PE?

Answer

1. Factors which predispose to development of pulmonary embolism are the presence of a deep venous thrombosis, a paralyzed and immobile leg, failure to prophylactically treat with LMW heparin, previous DVT or PE and other risk factors such as cancer.

Q11. How would you make the diagnosis of Pulmonary Thromboembolism?

Answers

1. Diagnosis is most often made with ventilation-perfusion scanning and/or spiral CT scanning.

Case Study (continued)

The patient has been treated with warfarin and during the visit you notice that he is both less alert and slower to respond than he has been during previous visits. The physiotherapist also mentions that he was very difficult to work with that day.

Q12. What would you be concerned with considering the patient is being anticoagulated?

Answer

1. Sudden change in a patient on anticoagulation is always a concern. Bleeding is the main complication and with decreased alertness an intracranial hemorrhage is a possibility.
2. A CT scan should be ordered to rule out intracranial bleeding.

Case Study (continued)

The CT shows a new small intracerebral haemorrhage. The warfarin is discontinued.

Q13. The resident asks you how long you should wait before restarting anticoagulation therapy after a warfarin-associated intracerebral haemorrhage.

Answer

1. Once the patient has stabilized consideration should be given to restarting the Warfarin, particularly if there is an ongoing risk of PE recurrence.
2. Decisions need to be made on a patient-by-patient basis. Factors which need to be taken into consideration are the general health of the patient, age, risk of falls and ongoing risk of PE recurrence.

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E7. Venous Thromboembolism Case Study

E7.1 Prophylaxis of Venous Thromboembolism Post Intracerebral Hemorrhage

Case Study

36 year old male admitted to stroke rehabilitation program and discharged 6 weeks later. 5 weeks previously he had suffered an intracerebral hemorrhage with right sided weakness. CT scan revealed a bleed in the left putamen and both caudate heads with compression of the ventricles. Etiology of the bleed was felt to be related to hypertension.

Q1. You know that the patient has a high risk for developing DVT, the nurse and the resident want to know if patients should be prophylactically anticoagulated after intracerebral hemorrhage?

Answer

The patient can be initiated on a typical LMW heparin prophylactic regimen without concern about increasing the size of the intracerebral hemorrhage.

Q2. Do you know of any other methods to prevent DVT in hemorrhagic strokes instead of pharmacological management?

Answer

There is moderate evidence that a combination of graded compression stockings and intermittent pneumatic devices reduce the risk of development of asymptomatic DVTs in patients with hemorrhagic stroke.

E7.2 Prevention of Recurrent Pulmonary Emboli in Intracerebral Hemorrhage

Case Study (continued)

Six days after presenting with an intracerebral hemorrhage, the patient was diagnosed with a symptomatic DVT and a symptomatic pulmonary embolus.

Q3. The patient has developed a PE in association with an intracerebral bleed. What do you do now?

Answer

This patient should not be anticoagulated but the risk of PE recurrence; it is necessary to insert a vena cava filter.

Case Study (continued)

An IVC filter was subsequently inserted. Anticoagulation was initiated 7 days later, almost 2 weeks post SAH. An attempt was subsequently made to remove the IVC filter; however, attempted retrieval of the IVC filter was unsuccessful because of the presence of a large embolism which was trapped in the filter. The filter was therefore left in place. On admission to rehabilitation he had been initiated onto anticoagulation, initially with heparin and quickly switched over to Coumadin after the filter was put into place.

E8. Post-Stroke Seizure Disorders

E8.1 Introduction and Case Study

Post stroke seizures may occur soon after stroke or be delayed; each appears to be associated with differing pathogeneses. Most seizures are single, either partial or generalized (Ferro and Pinto 2004. Wiebe and Butler (1998) noted that, “Seizures are the clinical expression of excessive, hypersynchronous discharge of neurons in the cerebral cortex.”

Whether seizures worsen outcomes remains unclear. Vernino et al. (2003) reported new-onset seizure among patients with ischemic stroke to be an independent risk factor for mortality on multivariate analysis (Relative risk 1.81; 95%CI 1.16-2.83). Bladin et al. (2000) also reported higher mortality among patients with seizures at 30 days and 1 year, compared to patients who were seizure free (25% vs. 7% and 38% vs. 16%). However, the authors did not control for the confounding effects of stroke severity or Comorbidity. The results of other studies have not supported an increased risk of mortality (Labovitz et al. 2001, Reith et al. 1997).

Case Study

A 55-year old man is admitted into the inpatient rehabilitation unit 12 days post-stroke. He had a cardioembolic stroke involving the territory of the left MCA, which is affecting the cortex. He is consequently being treated with warfarin. During the second night in the unit, the patient's partner advised the nurse that he was making strong repetitive movements with his right arm and thought he was having a seizure. When the nurse arrived, the patient had a marked tendency to fall sleep and he felt very tired.



Q1. What do you think caused this patient's episode and what risk factors may have contributed?

Answers

1. He likely suffered an epileptic seizure (the clinical expression of excessive, hypersynchronous discharge of neurons in the cerebral cortex) (Wiebe and Butler 1998).
2. Younger patients and men are at increased risk for seizure activity post stroke (Arboix et al. 1997, Giroud et al. 1994). A lesion involving the cerebral cortex is a prerequisite for the development of epilepsy (Olsen et al. 1987).

Q2. Would you describe this as epilepsy?

Answers

1. Epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures and by the neurobiologic, cognitive, psychological, and social consequences of this condition. The definition of epilepsy requires the occurrence of at least one seizure, although usually there needs to be more than one seizure.
2. One seizure in association with an enduring disturbance of the brain capable of giving rise to other seizures has been defined as epilepsy (Fisher et al. 2005).

E8.2 Incidence of Post-Stroke Seizures

Q3. Discuss the incidence of seizures post-stroke.

Answers

1. The incidence of seizures following ischemic or hemorrhagic stroke in earlier series is noted to be highly variable ranging from a low of 7.7% to a high of 42.8%. The average risk of seizure post-stroke is 10% within 9-10 years after stroke.
2. Hemorrhagic stroke patients have been found to have an almost 2-fold risk of developing a seizure following stroke compared to patients with an ischemic lesion (Bladin et al. 2000).

E8.3 Types and Timing of Post-Stroke Seizures

Q4. What are the most common types of seizures after stroke and what is the relation between seizures and time since the onset of stroke?

Answers

1. Black et al (1983) reported that 39% of seizures occurred within the first 24 hours, 57% within the first week and 88% within the first year.

2. The overall percentage after stroke of focal seizures was 50%, generalized seizures 32%, focal seizures with secondary generalization 15%, and complex partial seizures 2,5%. (Wiebe-Velazquez and Blume 1993).

E8.4 Treatment of Post-Stroke Seizures

Q5. Describe traditional treatment approach to post-stroke seizures.

Answer

1. There is consensus opinion that post-stroke seizures should be treated with anticonvulsant medication to prevent seizure recurrence.
2. Standard first-line therapy usually includes carbamazepine, valproic acid and phenytoin.
3. Phenytoin is known to interact with warfarin.
4. There is some concern that anti-epileptic drugs may impair recovery post stroke.
5. Benzodiazepines as an ongoing treatment should be avoided due to its sedating effects unless seizure activity is uncontrolled.

E8.5 Treatment of Status Epilepticus Post Stroke

Q6. The nurse asks you how you would treat status epilepticus (just in case).

Answers

1. Benzodiazepines are considered the best first-line drugs for managing status acutely with a seizure control rate of approximately 79%.
2. Both Lorazepam and Diazepam given intravenously are acute treatment strategies, although Lorazepam may be more effective in terminating status epilepticus (59-89% VS 43-76%) and has longer lasting anticonvulsivant properties (12 hours VS 20 Minutes for Diazepam).
3. Midazolam 10 mg given by the buccal and intranasal route is an alternative where intravenous medications are difficult to give.

E8.6 Impact of Anti-Epileptic Medications on Stroke Recovery

Q7. The patient's wife asks you if you need to treat the post-stroke seizures, that it seems to slow him down, and she is worried about side effects. What would you advise her?

Answers

1. There is some concern that the use of antiepileptic agents may impair recovery post stroke (Camilo and Golgstein 2004).
2. Benzodiazepines as ongoing treatment should be avoided unless seizure activity in uncontrolled due to its sedating effects.

3. There is consensus opinion that patients who have experienced seizures post stroke should be given preventive anticonvulsant medication to prevent reoccurrence.

E8.7 Phenytoin as a Treatment of Post-Stroke Seizures

Q8. The medical student asks you about treating post-stroke seizures with anticonvulsants. Is Phenytoin an appropriate treatment choice? What evidence is there for anticonvulsants?

Answers

1. Phenytoin is known to interact with warfarin; indeed, phenytoin interacts with many other drugs such as antibiotics.
2. There is no definitive evidence supporting one drug over another for the treatment of epilepsy in post-stroke patients. Standard first-line therapy usually includes carbamazepine, valproic acid, Lamotrigine and Phenytoin.
3. Gillad et al. (2007) showed that Lamotrigine may be better than Carbamazepine since Lamotrigine has relatively few side-effects, fewer potential drug interactions, and does not require blood monitoring in monotherapy.

E8.8 Driving and Post-Stroke Seizures

Q9. The patient's wife asks you about the possibility that her husband will be able to drive again. What can you tell her about epilepsy and driving?

Answer

1. The patient should be assessed by a neurologist and an EEG performed.
2. There needs to be an assumption he has suffered a single seizure event.
3. He will need to be seizure-free for at least 6 months before he can drive again, presuming the neurologist conducting the EEG concurs.
4. Individual circumstances may warrant prolonging or reducing the time period suggested.

Determining Medical Fitness to Operate Motor Vehicles: CMA Driver's Guide 7th edition, 2006

10.4 Seizures

As for all conditions, in all instances where a temporal recommendation is made, the time period should be considered a general guideline. Individual circumstances may warrant prolonging or reducing the time period suggested.

10.4.1 Single, unprovoked seizure before a diagnosis

Private drivers: These patients should not drive for *at least 3 months* and not before a complete neurologic evaluation — including electroencephalography (EEG) with waking and sleep recording and appropriate neurologic imaging, preferably magnetic resonance imaging (MRI) — has been carried out to determine the cause.

Commercial drivers: Commercial drivers should be told to stop driving all classes of vehicles at once. For these drivers, there is a need for even greater certainty that another seizure will not occur while they are driving. As a minimum, commercial drivers should follow the private driver guideline and not drive private vehicles for at least 3 months after a single, unprovoked seizure. If a complete neurologic evaluation, including waking and sleep EEG and appropriate neurologic imaging, preferably MRI, does not suggest a diagnosis of epilepsy or some other condition that precludes driving, it is safe to recommend a return to commercial driving after the patient has been seizure free for 12 months.

10.4.2 After a diagnosis of epilepsy

Patients may drive any class of vehicle if they have been seizure free for 5 years with or without anticonvulsive medication.

Private drivers: Patients with epilepsy who are taking anti-seizure medication should not be recommended for Class 5 or 6 licensing until the following conditions are met:

- **Seizure-free period:** The patient should be seizure free on medication for not less than 6 months, unless seizures with altered awareness have occurred more than once a year in the previous 2 years, in which case the seizure-free interval should be 12 months. With certain types of epilepsy, this period may be reduced to not less than 3 months on the recommendation of a neurologist, stating the reasons for this recommendation. The seizure-free period is necessary to establish a drug level that prevents further seizures without side effects that could affect the patient's ability to drive safely. The anti-seizure medication should have no evident effect on alertness or muscular coordination.
- **Patient compliance with medication and instructions:** The attending physician should feel confident that the patient is conscientious and reliable and will continue to take the prescribed anti-seizure medication as directed, carefully follow the physician's instructions and promptly report any further seizures. Medication compliance and dose appropriateness should be documented with drug levels whenever reasonably possible.

Physicians should advise epileptic patients that they should not drive for long hours without rest or when fatigued. Patients who require anti-seizure medication and who are known to drink alcohol to excess should not drive until they have been alcohol and seizure free for at least 6 months. These patients often neglect to take their medication while drinking. As well, alcohol withdrawal is known to precipitate seizures and the use of even moderate amounts of alcohol may lead to greater impairment in the presence of anti-seizure medication. Patients taking these drugs should be advised not to consume more than 1 unit of alcohol per 24 hours.

A patient who stops taking anti-seizure medication against medical advice should not be recommended for driving. This prohibition on driving may change if the physician feels confident that the formerly noncompliant patient, who is again taking anti-seizure medication as prescribed, will conscientiously do so in the future and if compliance is corroborated by therapeutic drug levels, when available.

Commercial drivers: It can be unsafe for commercial drivers who must take anti-seizure medication to operate passenger-carrying or commercial transport vehicles (Classes 1–4). For these drivers, there is a need for even greater certainty that another seizure will not occur while they are driving. Commercial drivers are often unable to avoid driving for long periods of time, frequently under extremely adverse conditions or in highly stressful and fatiguing situations that could precipitate another seizure. Unfortunately, seizures do sometimes recur even after many years of successful treatment.

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E9. Central Pain State

E9.1 The Incidence of Central Pain Post Stroke

Q1. What is the incidence of central pain post stroke?

Answer

1. Central post-stroke pain occurs in less than 2% of stroke patients.

E9.2 Pathophysiology of Central Pain Post Stroke

Q2. What is the pathophysiology of central pain post stroke?

Answers

1. The pathophysiology of central pain post stroke remains largely unknown.
2. Must be damage to the spino-thalamo-cortical pathway with disturbance in temperature and pain sensation.
3. Damage to the spinothalamic tract resulting in denervation hyperexcitability of cortical or thalamic neurons are currently the most popular hypothesis for the pain.
4. Spontaneous or evoked dysesthesia, allodynia/hyperalgesia are manifestations of central post-stroke pain; paradoxical allodynia with associated numbness in the same territory.

Case Study

A 46 year old male sustained a right subcortical hemorrhage. He subsequently experienced gradual improvement in his motor function so that he was again ambulatory without support. However, since about one month after his stroke he had been troubled by a painful "pins and needles" sensation involving his entire left side, except for his scalp. He also had a burning pain ("like a sunburn") without superimposed shooting or lancinating pains. He was more comfortable at rest with a reported pain intensity of about 4/10. He had extreme sensitivity to light touch and other stimulation involving the left side of his body and in particular, just rubbing of his clothes while walking exacerbated his pain significantly. On neurological examination his cranial nerves were intact, motor tone was normal although there was a left sided pronator drift and mild weakness in left hip flexion. Fine finger coordination was normal bilaterally. On sensory examination there was blunting to pinprick involving his entire left side, except for his face. He had marked touch evoked pain or allodynia on the left side. The cold tuning fork was perceived as being even colder on the left side. Vibration sense was impaired in the left fingers and toes and he had

difficulty detecting fine excursion of the left toes. Cortical sensation was intact and in particular, there was no sensory neglect. Gait testing revealed less arm swing on the left which was directly related to the fact that stimulation from his clothes increased his pain.

E9.3 Defining Post-Stroke Central Pain States

Q3. Define and Describe the Post-Stroke Central Pain State.

Answer

1. Central pain is pain due as a direct consequence of the stroke.
2. Described as burning or unpleasant sensations (parasthesiae) made worse with physical movements, emotional stress, cold and light touch.
3. Associated sensory abnormality on affected side.

Q4. Define the terms “dysesthesia”, “allodynia”, and “hyperalgesia”.

Answers

1. *Dysesthesia*: Unpleasant sensations, either spontaneous or evoked (Andersen et al. 1995).
2. *Allodynia*: Abnormally unpleasant somatosensory experience, often poorly localized, elucidated by normally non-nociceptive stimuli (Andersen et al. 1995).
3. *Hyperalgesia*: Increased pain response to a normally painful stimulus (Andersen et al. 1995).

E9.4 Treatment of Central Post-Stroke Pain

Q5. Describe an algorithm treatment approach to Central Post Stroke Pain.

Answer

1. Majority of CPSP are intractable to therapeutic interventions.
2. First line treatments include tricyclic antidepressants and antiepileptics.
3. Second line treatment is opioids.

Case Study (continued)

When seen almost 18 months later, he had been tried on tricyclic antidepressants without benefit. He continued on Tegretol 100 mg BID which was the maximum dose he was able

to tolerate, He also took Tylenol #2 x2 tablets up to 4 times daily with minimal relief. He was Lorazepam one tablet at night.

Q6. What options would be available now?

Answer

1. Second-line treatment would include stronger narcotic analgesics such as Oxycodone (short-acting or long-acting) or Morphine (long-acting).
2. Alternative anti-epileptics such as Dilantin, Gabapentin and Pregablin.

Case Study (continued)

The patient was initiated on a stronger analgesic Percocet 2 tablets qid and Neurontin (Gabapentin), gradually increasing dose to 300 mgs qid as well as Venlafaxine 75 mg daily. Duragesic was initiated and increased to 50 mcg per hour but was only receiving pain relief for the first 2 of 3 days. He experienced moderate but still inadequate pain relief. His Neurontin was increased to 1,600 mg tid without pain relief and Dilantin 350 mg daily was added to deal with seizures. Duragesic was increased to 150 mcg per hour every 2 days. Methadone was used to replace the Duragesic at a rate of 20 mg tid which was increased to 50 mg q6h. Neurontin was decreased and Lyrica initiated at 300 mg bid; Venlafaxine was increased to 150 mg daily while Nabilone was given at 1mg daily.

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E10. Urinary Incontinence

Canadian Stroke Strategy Guidelines: Recommendation 4.2d – Continence

- i. All stroke patients should be screened for urinary incontinence and retention (with or without overflow), fecal incontinence and constipation [Evidence Level C] (RNAO).
- ii. Stroke patients with urinary incontinence should be assessed by trained personnel using a structured functional assessment [Evidence Level B] (AU).
- iii. The use of indwelling catheters should be avoided. If used, indwelling catheters should be assessed daily and removed as soon as possible [Evidence Level C] (AU, CSQCS, RCP, VA/DoD).
- iv. A bladder training program should be implemented in patients who are incontinent of urine [Evidence Level C] (AU, VA/DoD).
- v. The use of portable ultrasound is recommended as the preferred noninvasive painless method for assessing post-void residual and eliminates the risk of introducing urinary infection or causing urethral trauma by catheterization [Evidence Level C] (CCF).

E10.1 Normal Bladder Functioning

Q1. Describe the various areas of the central nervous system and peripheral nervous systems involved in the storage and emptying of urine.

Answers

Normal Bladder (detrusor) and urethral functioning involves the following areas of the central and peripheral nervous systems (Borrie1998):

1. The sympathetic nervous system relaxes the detrusor muscle while internal urethral sphincter control is maintained by sympathetic alphaadrenoreceptors.
2. Parasympathetic acetylcholine receptors mediate detrusor contracture.
3. Somatic (voluntary) nervous system innervates the pelvic floor muscles, including the external urethral sphincter.
4. A micturition centre in the brainstem (pons) informs when the bladder is filling and controls the sacral reflex when bladder filling reaches a certain level.
5. The micturition centre in the frontal lobes provides conscious input to the pontine micturition centre allowing the inhibition of urination until the time of voluntary control.

E10.2 Urinary Dysfunction Post Stroke

Q2. Describe the different types of urinary dysfunction occur post-stroke.

Answers

1. The most frequently occurring voiding abnormalities associated with a stroke have been identified as urinary frequency, urge incontinence and urinary retention (Marinkovic and Badlani 2001).
2. Urinary tract infection is not uncommon in stroke patients.

Q3. What three mechanisms are thought to be responsible for urinary incontinence post stroke?**Answers**

1. Detrusor hyperreflexia. Urge incontinence and bladder hyperreflexia from damage to neuromicturition pathways.
2. Bladder retention. Overflow incontinence related to diabetic neuropathy or medications.
3. Stroke related barriers: paresis, cognition and language deficits.

E10.3 Detrusor Hyperreflexia Post Stroke**Q4. Discuss detrusor hyperreflexia.****Answer**

Hyperactive bladder empties suddenly at a usually lower than normal volume.

E10.4 Urinary Retention Post Stroke**Q5. Discuss urinary retention and potential contributing factors.****Answer**

1. Urinary retention is common following an acute stroke (21-47%).
2. Potential contributing factors include difficulty with communication, mobility and decreased consciousness.
3. Other contributing factors may be a hyperreflexic bladder, as seen in diabetic neuropathy, or an obstruction such as prostatic hypertrophy.
4. Occasionally urinary retention will persist in very severe stroke patients, often in patients with embolic strokes with no previous warnings.

E10.5 Other Factors Contributing to Post-Stroke Incontinence

Q6. Discuss Other Factors Contributing to Post-Stroke Incontinence.

Answers

Stroke results in a number of factors which can affect continence, but which are often overlooked:

1. Communication difficulties, particularly an inability to communicate voiding needs, either due to aphasia, dysarthria or confusion/cognitive impairments.
2. Mobility problems, such as hemiplegia make some patients dependent on caregivers to void in a socially appropriate manner. Lack of caregiver support may also make it difficult to toilet the stroke patients quickly enough.
3. Post stroke depression and confusion may result in a failure to communicate the need for assistance.
4. Medications, such as diuretics can increase the frequency of the need to void; others can increase confusion, while still others such as antihypertensives may affect the autonomic nervous system leading to retention.

E10.6 Course of Urinary Incontinence Post Stroke

Q7. How common is urinary incontinence following stroke? What is the natural history?

Answer

1. Urinary incontinence has been reported in 37-79% of stroke patients in the acute phase and 17% of stroke survivors in the community.
2. Most incontinence resolves without treatment over 8 weeks although a significant percentage (14-19%) still have incontinence at 6 months.

E10.7 Risk Factors for Urinary Incontinence Post Stroke

Q8. What are the risk factors for urinary incontinence post stroke?

Answers

1. Severe strokes have a higher incidence of urinary incontinence.
2. Premorbid history of urinary incontinence, i.e comorbidities.
3. Motor weakness and mobility difficulties, including ataxia.
4. Altered level of consciousness.
5. Cognitive impairment
6. Depression
7. Elderly
8. Diabetic

E10.8 Consequences of Urinary Incontinence Post Stroke

Q9. Discuss the relationship between urinary incontinence and institutionalization.

Answers

Stroke survivors with persistent urinary incontinence tend to have greater disability, a poor prognosis, greater morbidity and mortality during their hospital stay and are more likely to be institutionalized.

E10.9 Urinary Tract Infection Post Stroke

Q10. How common are UTIs Post Stroke and what are risk factors for UTI?

Answers

1. UTI is the most common medical complication in stroke rehabilitation.
2. Risk factors include use of beta-blockers and high post-void residuals.

E10.10 Post-Void Residual Urine Testing

Q11. Discuss the importance of the post-void residual in diagnosis of bladder dysfunction post stroke.

Answer

1. PVR is able to determine if bladder emptying is complete.
2. PVR > 150 cc is generally regarded as abnormal.
3. Increased PVRs increases the risk of urinary tract infections.

E10.11 Diagnosis of Bladder Dysfunction

Q12. Provide a classification of bladder dysfunction post stroke.

Answers

1. Detrusor instability or hyperreflexia with urge incontinence.
2. Stress incontinence.
3. Overflow incontinence with incomplete emptying.
4. Mixed types of incontinence.
5. Functional incontinence, due to nonurinary factors.
6. Iatrogenic incontinence, due to drugs or restraints.

E10.12 Assessment and Management of Urinary Incontinence Post Stroke

Q13. Describe an algorithm for the assessment and management of urinary incontinence.

Answer

1. History of symptoms to determine which type of incontinence the patient suffers from (i.e., urge, stress, mixed, overflow, or functional).
2. Further assessment may include a physical examination as well as urodynamic investigations (such as a postvoid residual test or cystometry).

E10.13 Treatment of Post-Stroke Urinary Dysfunction

Q14. What are the different treatment options for post-stroke urinary dysfunction?

Answers

1. Interventions used to treat UI depends on type of incontinence.
2. Initially behavioural interventions with scheduled voiding, pelvic floor exercises and biofeedback.
3. Pharmacological interventions
4. Catheterization.

E10.14 Behavioural Interventions for Incontinence Post Stroke

Q15. Discuss behavioural interventions for incontinence post stroke.

Answer

1. Scheduled voiding q 2-4 hours
2. Fluid restriction.

E10.15 Pharmacological Interventions for Incontinence Post Stroke

Q16. Discuss pharmacological interventions for incontinence post stroke.

Answer

1. Pharmacological treatment will depend on the cause of the stroke.
2. The vast majority of bladder incontinence is due to detrusor hyperreflexia due to central loss of inhibition.
3. Drugs with anticholinergic actions are recommended in these cases, i.e Oxybutynin.
4. For overflow incontinence due to detrusor inactivity, a cholinergic such as Bethanecol is recommended.

5. For overflow incontinence due to outlet obstruction, an alpha-adrenergic blocker may be recommended.

E10.16 Catheters for Bladder Dysfunction Post Stroke

Q17. Discuss the use of catheters for bladder dysfunction post stroke.

Answer

1. Catheters for bladder dysfunction should always be seen as a treatment of last resort.
2. Intermittent catheterization is the preferred option in urinary incontinence with overflow incontinence.
3. An indwelling catheter should be limited to those patients with intractable urinary retention, continuous wetness (+/- skin breakdown) and those who have need of monitoring.

E10.17 Management of Bladder Retention

Q18. Describe an algorithm for the management of bladder retention?

Answer

1. Look for reversible causes of bladder retention and correct it.
2. If there is no reversible cause, need to perform urodynamic studies to determine whether it is a sphincter problem (not opening) or a detrusor problem (hypoactive bladder).
3. If sphincter problem consider alpha-blocker medications; if not successful consider botulinum toxin into the sphincter.
4. If detrusor problem consider cholinergic medication such as Bethanecol.
5. Once voiding begins monitor with post-void residuals.
6. If not successful catheterization is necessary; many stroke patients cannot do intermittent catheterizations which may require an indwelling catheter.

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