

E. Medical Complications

Robert Teasell MD FRCPC, Manuel Murie-Fernandez MD, Andrew McClure, Norine Foley

E1. Dysphagia.....	2
E2. Dysphagia Case Study.....	4
E3. Dysphagia Case Study.....	6
E4. Dysphagia in a Nursing Home Stroke Patient.....	11
E5. Nutritional Issues Following Stroke.....	12
E6. Deep Venous Thromboembolism.....	14
E7. Venous Thromboembolism Case Study.....	18
E8. Post-Stroke Seizure Disorders.....	20
E9. Central Pain State.....	24
E10. Urinary Incontinence.....	26

29 Pages

E1. Dysphagia

E1.1 Introduction to Dysphagia Post Stroke

Q1. Define dysphagia.

Q2. Why is dysphagia important following a stroke?

E1.2 The Normal Swallowing Process

Q3. Describe the normal swallowing process.

E1.3 Defining Dysphagia and Aspiration

Q4. What is the difference between Dysphagia and Aspiration?

E1.4 Risk Factors for Aspiration Post Stroke

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

E1.5 Dysphagia Post Stroke

Q6. How common is dysphagia following acute stroke?

E1.6 Silent Aspiration

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

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

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?

E1.8 Risk Factors for Aspiration Pneumonia

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

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)?

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?

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.

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.

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.

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.

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)?

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?

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 demonstrated 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 recommend 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 subsequent 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?

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?**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?

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?

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?

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)?

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?

Q3. What is this woman's nutritional status?

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

Q4-1. Describe the Subjective Global Assessment.

Q4-2. Describe the Mini-Nutritional Assessment.

E5.3 Protein and Energy Requirements

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

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

Q7. How do you monitor and assess the progress?

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?

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.

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?

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.

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

E6.4 Prevention of Deep Venous Thrombosis Post Stroke

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

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

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

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?

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

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

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?

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.

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?

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

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?

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?

Q2. Would you describe this as epilepsy?

E8.2 Incidence of Post-Stroke Seizures

Q3. Discuss the incidence of seizures post-stroke.

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?

E8.4 Treatment of Post-Stroke Seizures

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

E8.5 Treatment of Status Epilepticus Post Stroke

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

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?

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?

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?

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.

E9. Central Pain State

E9.1 The Incidence of Central Pain Post Stroke

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

E9.2 Pathophysiology of Central Pain Post Stroke

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

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.

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

E9.4 Treatment of Central Post-Stroke Pain

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

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?

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.

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.

E10.2 Urinary Dysfunction Post Stroke

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

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

E10.3 Detrusor Hyperreflexia Post Stroke

Q4. Discuss detrusor hyperreflexia.

E10.4 Urinary Retention Post Stroke

Q5. Discuss urinary retention and potential contributing factors.

E10.5 Other Factors Contributing to Post-Stroke Incontinence

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

E10.6 Course of Urinary Incontinence Post Stroke

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

E10.7 Risk Factors for Urinary Incontinence Post Stroke

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

E10.8 Consequences of Urinary Incontinence Post Stroke

Q9. Discuss the relationship between urinary incontinence and institutionalization.

E10.9 Urinary Tract Infection Post Stroke

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

E10.10 Post-Void Residual Urine Testing

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

E10.11 Diagnosis of Bladder Dysfunction

Q12. Provide a classification of bladder dysfunction post stroke.

E10.12 Assessment and Management of Urinary Incontinence Post Stroke

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

E10.13 Treatment of Post-Stroke Urinary Dysfunction

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

E10.14 Behavioural Interventions for Incontinence Post Stroke

Q15. Discuss behavioural interventions for incontinence post stroke.

E10.15 Pharmacological Interventions for Incontinence Post Stroke

Q16. Discuss pharmacological interventions for incontinence post stroke.

E10.16 Catheters for Bladder Dysfunction Post Stroke

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

E10.17 Management of Bladder Retention

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