



NRS 221

College Laboratory Guide

Fall 2014

Student Name _____

Week 1

Concept: Safety

Lab Activities

- Fulmer SPICES: An Overall Assessment Tool for Older Adults

http://consultgerirn.org/resources/media/?vid_id=4200873#player_container

- Mini-Cog

http://consultgerirn.org/resources/media/?vid_id=4361918#player_container

- Geriatric Depression Scale

http://consultgerirn.org/resources/media/?vid_id=4200933#player_container

- Role Playing- Personally experiencing the challenges of activities of daily living when one has alterations of bodily functions. (discussion/teaching plan)

- Assessing for Elder Abuse

http://consultgerirn.org/resources/media/?vid_id=5004146#player_container

- Utilize SBAR for handoff communication , case scenario challenges of suspected elder abuse

Math

1. A patient is ordered Aricept (donepezil HCl) 7.5 mg po at HS. On hand you have 5 mg tablets. How many tablets will you give for the correct dose?
2. The order reads Prozac (fluoxetine hydrochloride) liquid 10 mg po daily. The medication solution reads Prozac liquid 20 mg per 5 mL. How much will you measure for the correct dose?
3. The patient is to receive 1000 mg of carafate (sucralfate) po 30 minutes prior to their meals. On hand you have 1 tablet = 1 gram. How many tablets will you give for the correct dose?
4. A patient is to receive 150 mL of D5/W to infuse in 1 hour. The tubing you have is calibrated to 15gtt/mL. Calculate the gtt/min.
5. A patient is ordered Toradol (Ketorolac tromethamine) 60 mg IM stat. What you have available is toradol 30 mg/mL. What will you draw up as the correct dose?
6. An adult patient who weighs 150 lbs. is ordered Gentamicin sulfate 1.5 mg/kg IV 30 minutes prior to a procedure. What dose will you draw up?

Week 2

Concepts: Sensory Perception, Safety

Lab Activities

- Cranial Nerve Assessment
https://meded.ucsd.edu/clinicalmed/pe_cranialexam.pdf
- Appropriate method to instill ophthalmic medications to pediatric and adult patients.
<https://www.youtube.com/watch?v=SnAfc6h4ax4>

<http://www.healthychildren.org/English/safety-prevention/at-home/medication-safety/Pages/How-to-Give-Eye-Drops-and-Eye-Ointment.aspx>
- Appropriate method to instill otic medications to pediatric and adult patients.

<https://www.youtube.com/watch?v=P5WCj33VBGg>

<http://www.healthychildren.org/English/safety-prevention/at-home/medication-safety/Pages/How-to-Give-Ear-Drops.aspx>
- Assessing visual acuity

<https://www.youtube.com/watch?v=NCTxxcxtcHs>
- Assessing hearing acuity

https://www.youtube.com/watch?v=LR_pywMtv9c
- Removing and Cleaning an artificial eye

<https://www.youtube.com/watch?v=80YHi-qAhhg>
- Removing & cleaning contact lenses

<https://www.youtube.com/watch?v=OrQ9nr9lQzY>

- Removing, Cleaning& inserting a hearing aid

<https://www.youtube.com/watch?v=ie7vm881C1w>

https://www.youtube.com/watch?v=3py_wqaUFwA

- Role Play: Develop a teaching plan for parents of an 18 month old toddler who will be receiving nonsurgical treatment for strabismus, utilize SBAR for handoff communication.

Math

1. The order is prednisone 50 mg po now. You have on hand prednisone 20 mg tablets. How many should you give?
2. The order reads pilocarpine hydrochloride 0.25% ophthalmic solution 1 gtt to OU at 0900 and 2100. Describe how you will administer the 0900 dose.
3. The physician orders Vitamin B1 (thiamine hydrochloride) 10 mg IM bid. You have a 10mL multiple dose vial of Vitamin B1 (thiamine HCL) of 100 mg/mL. How much will you give as a dose?
4. The order reads vancomycin HCL 0.5 g po bid. On hand you have a 100 mL bottle of vancomycin HCL liquid, 125mg/5mL. How much will you pour as one dose?

5. IV order of 1 L D5/W to infuse in 24 hours. The tubing drop factor is 10 gtt/mL. Calculate how many drops per minute you would have to manually adjust the drip rate.

6. You have an infusion pump which is calibrated to infuse mL/hr. Using the above order, at what rate would you set the infusion pump?

Week 3

Concepts: Mobility, intracranial regulation, safety

Lab Activities

- Video on Glasgow Coma Scale
<https://www.youtube.com/watch?v=T93Ah9ZkurI>
- Role Play: Perform a Glasgow Coma Scale assessment
- NPSG hand off report, use of SBAR to report your findings.
- HESI Case Study “Seizure Disorder”. Patient name Alanna Milstein
- Implementing Seizure Precautions
- Develop educational plans for a person with Parkinson Disease at the various stages.

- Develop a plan of care for a child with Cerebral Palsy (and their family).

Math

1. Order is levodopa 0.75 g po daily. On hand you have 250 mg tablets. How many tablets will you give?
2. Order is dilantin (phenytoin) suspension 150mg po bid. On hand is a bottle containing dilantin 75mg/7.5 mL. How many mL will you give for one dose?
3. The physician orders gentamicin 24 mg IVPB q8 hr for a child weighing 26 lbs. The suggested range is 2-2.5 mg/kg every 8 hrs.
 - a. What is the suggested dosage range for this child?
 - b. Is this a safe dose for this child?
4. The physician orders ampicillin 50mg/kg/day to be divided into 4 doses. The patient weighs 20 kg. How many mg would be in each dose?
5. Order is Ancef 50mg IVPB in 100 mL NS to infuse in 30 mins. The infusion pump delivers mL/hr. At what rate will you set the infusion pump?

Week 4

Concepts: Mobility, intracranial regulation, safety

Lab Activity

- Stroke/Brain Attack DVD (view and discuss)
- Review Post Stroke Depression (journal article /questions)
- History and physical exam for suspected CVA (role play)
- NIH Stroke Scale Testing
- Assisting with pediatric & adult lumbar puncture

Math

1. Order is for heparin 7,000 units subQ Q 12 hr. On hand you have a vial containing 5,000 u/mL. How much will you draw up in the syringe for the dose?
2. Calculate the rate of infusion for a 1000 mL of D5/W over 12 hours.
3. Order is Benadryl 75 mg po now. On hand you have Benadryl 25mg/5mL. How much will you give for this dose?

4. Order is to infuse a solution of heparin 20,000U to 1 L of D5/W at 80 mL/hr. Calculate the dose of infusion.

Week 5

Concept: Safety

Lab Activities:

- Establishing IV Infusions
- Using IV Infusion Pumps
<https://www.youtube.com/watch?v=dCyakRWH9a0>
- Patient Safety (NPSG & QSEN) Case Study
- Josie King Story
<https://www.youtube.com/watch?v=JeVcXhvPvbU&feature=youtu.be>
https://www.youtube.com/watch?v=ak_5X66V5Ms

Lab Resources:

- National Patient Safety Goals website:
http://www.jointcommission.org/standards_information/npsgs.aspx
- Speak Up Campaign
<http://www.jointcommission.org/speakup.aspx>
- How to report concerns and file a complaint:
http://www.jointcommission.org/report_a_complaint.aspx
- QSEN website:
<http://qsen.org/>

Post Stroke Depression Article

Early assessment and interventions can promote optimal recovery.

By Susan Fralick-Ball, PsyD, MSN, CH

The goal this article is to provide nurses with current information on post-stroke depression. After reading this article, you should be able to:

1. Describe post-stroke depression as a complication of ischemic stroke.
2. Compare the two forms of post-stroke depression.
3. Discuss the care plan for a patient with a stroke complicated by depression.

As sequelae to a right-sided or left-sided (ischemic) stroke, occurring in both the young and old, post-stroke depression (PSD) is a real and present danger to optimal stroke recovery. In the U.S., there are more than 700,000 strokes annually, at a rehabilitation cost of more than \$58 billion. Making the most of full physical and mental health recovery needs to be at the core of stroke treatment.

PSD remains recognized, yet under-diagnosed and under-treated. The medical community is attempting to focus on the determinants of PSD during the acute and chronic phases of stroke. We are looking for early predictors or risk factors to depression to address early intervention strategies. Many in our population do not know or recognize the signs of stroke or depression in themselves or others, thus possibly leading to overwhelmingly negative outcomes of brain events.

Various experts agree there are two forms of PSD: major endogenous, psychotic depression and minor, dysthymic reactive depression. With endogenous PSD, the monoamine pathways linking the brain stem to the cortex -- and frontal lobe executive functioning -- have been interrupted. Ten to 50 percent of stroke survivors experience this form of major PSD. Generally, there is full recovery from the depression in 1-2 years following the traumatic stroke event, yet 7-10 percent are associated with post-stroke suicide ideation.

Dysthymic reactive depression has no connection to brain lesion locus and becomes apparent in 15-40 percent of post-stroke clients (prevalence higher in females over males), with varying degrees of recovery over the subsequent 2 years. If depression reduction does not occur within

the first few years, the minor form of PSD can become a major depressive disorder, necessitating longer-term medical and psychological treatment to help alleviate the symptoms of this devastating form of depression. Despite significant worldwide research, there is no agreement on the causal mechanisms, risk factors or consequences of PSD; evidence supports a multifactorial origin.

Symptoms

Typical symptoms of depression include a sense of hopelessness that disrupts one's ability to function, sleep disturbances, radical changes in eating patterns, lethargy, social withdrawal, irritability, fatigue and lowered self-esteem that leads to the sense of uselessness. Suicidal thoughts may ensue. In PSD, these signs can result from vascular changes and psychological reactions to disability and often are provoked by the lesion.

The neurophysiology of stroke indicates a probable disruption of amine-containing pathways by stroke lesion. Noradrenergic and serotonergic cell bodies in the brain stem (locus coeruleus and midline raphe nucleus of lower pons and medulla, respectively) send messages through the medial forebrain to frontal cortex for interpretation and executive orchestration.

Depression may be caused by depletion of total available norepinephrine and/or serotonin, or interference with receptor sites. Agitation, confusion, tremor, myoclonus and hyperthermia may result as a serotonin syndrome emerges post-stroke. Serotonin syndrome is a triad of rapid-onset cognitive, autonomic and somatic toxic effects ranging from mild to life-threatening after administration of therapeutic dosages of serotonin and/or concomitant medications.

Signs and symptoms include mental confusion, hypomania, hallucinations, agitation, headache, coma, shivering, sweating, hyperthermia ($>104^{\circ}$ F), hypertension, tachycardia, nausea, diarrhea, muscle twitching, hyperreflexia and tremor. Other manifestations include metabolic acidosis, rhabdomyolysis, seizures, renal failure and disseminated intravascular coagulation (DIC) secondary to the hyperthermia.

Serotonin antagonists and stoppage of the serotonin medication must be instituted immediately when this syndrome occurs. Benzodiazepines are indicated to control agitation. Antipyretics are not indicated since the increased body temperature is due to muscular activity, not a temperature

set-point abnormality. Direct-acting sympathomimetics (e.g., epinephrine, etc.) may be needed to counter the autonomic symptoms. Serotonin syndrome differs from neuroleptic malignant syndrome in subtle ways, yet classic differential includes hyperkinesias and clonus for serotonin syndrome and bradykinesia with "lead pipe" rigidity for neuroleptic malignant syndrome.

Lasting Effects

Severe PSD may eventually involve physiological changes, e.g., diurnal mood variation, early morning waking and loss of appetite. Untreated PSD can lead to reduction in quality of life, poorer prognosis and increased mortality, especially for those living in communal vs. home situations following stroke.

Differences in the presentation of PSD appear when considering right-sided vs. left-sided lesions. The lasting psychological effects following a brain event in the right hemisphere include significant variance from the person's normal emotional output and relative lack of control. Many of the lesions to the right frontal and parietal lobes leave impairments for recognizing emotions expressed through tone of voice, identification of facial expressions, and difficulty expressing emotion through facial movements, as well as anhedonia (severe lack of interest in people/interests).

Cerebral ischemia leads to decrease in acetylcholine release, which may cause many of the post-stroke behavioral changes. Mania and hypomanic reactive responses are more common with right-sided vs. left-sided lesions.

Patients with right-sided stroke lesions become desocialized. This often is the beginning of strain on social contact and communication. A family history of psychiatric disorders or having had a previous left-sided stroke increases the risk of PSD in right-sided stroke patients.

Left-sided lesions in the frontal or basal ganglia areas (putamen and/or caudate nucleus) often result in major depression. An increased catastrophic response is thought to be due to an inability to up-regulate serotonin receptors since serotonin binding is greater on the right. Vascular lesions also may interfere with serotonin transduction throughout the brain.

It also is possible to classify depressive symptoms post-stroke into direct and indirect cause-and-effect responses. Indicators for reactional depression include:

- aphasia;
- amnesia and cognitive impairments;
- anosognosia (denial of disability);
- denial of depressive signs;
- aprosodia (poor speech comprehension);
- catastrophic reaction;
- neurological apathy syndromes (e.g., frontal lobe syndrome);
- dementia.

Often, the signs of PSD are clinically similar to the depression associated with dementia of the Alzheimer's type.

Indirect symptoms are linked to factors common to many severely ill, hospitalized or stroke patients: controlled appetite due to tube feedings or dysphagia, frequent awakenings with a total poor sleep pattern, significant time in bed or being relatively immobile, delirium or speech disruptions. Many of these post-stroke outcomes severely hamper the effectiveness of physical, occupational, speech/language and psychological therapies.

PSD related to lesion location can be characterized by the following:

- Bilateral lesions in the anterior frontal and temporal lobes and caudate nucleus are associated with increased risk of PSD.
- Left basal ganglia lesions at the head of dorsolateral caudate result in apathy and poor initiation.
- Incidence and severity of PSD increase with close proximity to left frontal pole lesions.
- Emotion-related processing impairments are greater with right hemisphere and frontal lobe lesions, since they serve emotional communication.

- Frontal lobe anterior and middle cerebral artery lesions result in apathy.
- Left anterior subcortical lesions show greater incidence of PSD than on the right side.
- When lesions are within 40 percent of either frontal pole, the PSD rate is 60 percent or greater.

Cerebrovascular disease also is found concomitantly with cardiovascular disease. When adding the co-condition of depression, the mood disorder is found both independently and additively when both heart disease and brain injury occur.

For many years, experts have recognized the incidence of depression following myocardial infarction, cardiac valvular disease, hypertension and dysrhythmias. Depression also remains an enduring predictor of post-cardiac incident morbidity and mortality, often severely affecting rehabilitation, return to sexual function and adherence to therapeutic regimen. With the advent of selective serotonin reuptake inhibitors (SSRIs) for the treatment of depression, there has been increased use of this treatment that features relatively few cardiac side effects.

Assessment of PSD

Diagnosis of PSD can be made with a combination of psychiatric interview, interviewer-administrated scales and self-rated depression scales (Aphasic Depression Scale, Beck Depression Inventory, Geriatric Depression Scale, Hamilton Depression Rating Scale and others), and the use of the criteria from the Diagnostic and Statistical Manual-IV revision. Depending on the scale utilized, many physical accompany symptoms also can be elucidated while screening for depression post-stroke. Other symptoms to consider include apathy, blunted affect, irritability, mania, fatigue, sleeping disorders and the like. A Post-Depression Rating Scale was developed specifically to identify depression in stroke survivors.

Additional losses of family cohesiveness, job, functional ability, cognitive clarity, speech recognition and production need to be assessed. Researchers of longitudinal studies have concluded that stroke and/or depressive symptoms at baseline evaluation predict clinically significant depression into the future. However, predictors of PSD change over time, especially if the stroke has involved expressive-communication impairments. Physical disability and cognitive impairment were consistently associated with depression. Since the negative impact of mood on physical recovery and reintegration is so prevalent, physicians and treatment team members are becoming more educated and likely to treat stroke survivors earlier.

Women, Stress & PSD

Since women have become an integral part of the out-of-home workforce and subject to similar working stress and pressures men have encountered, women's stress and numbers of cardiac and cerebral vascular insults have significantly increased. The rise of metabolic syndrome (high blood pressure, diabetes, central-body weight gain and altered lipid profile) during and after menopause with the loss of endogenous estrogens, and the occurrence of atrial fibrillation in women are major contributors to brain incidents.

Additionally, women's response to stress and PSD differs from men's. Women carry a higher stroke burden than the men, both because women have a longer life expectancy -- and strokes occur at late ages for many women -- and because most stroke deaths occur in women. Women ages 45-54, while continuing to be active mothers, employees, spouses, possible grandmothers and/or caretakers for older parents, are twice as likely as men in the same age bracket to have strokes.

Differences between the sexes in relation to stroke are increasingly being recognized. For example, among stroke survivors, women tend to have worse outcomes than men due to more-severe pre- and post-stroke disability, and an increased likelihood of post-stroke institutionalization and loss of independence for women.

Being female raises more questions regarding the occurrence of PSD:

- Is pre-stroke depression already in place (with depression nearly twice as prevalent in women than men)?
- Has there been an extremely erratic or sudden loss of estrogen during menopause?
- During menopause, have sleep issues contributed to depression?
- Have general social issues and stress contributed to depression?

Since female communication and language systems in the brain are more cross-hemispherically connected, they often suffer significant speech/language deficits during stroke, resulting in reactive PSD. Women also have been lax in noting and reporting initial symptoms of stroke when emergency treatment can be initiated to limit the type and amount of stroke sequelae.

Psychotherapy

Treatment of PSD includes psychotherapy, medication and time from insult. As of our current assessment, only one-third to less than one-half of all post-stroke patients are adequately treated for depression.

Assessment of symptoms is problematic since there is no gold standard for diagnosis after recent stroke; however, rating scales are available and help with initial and ongoing evaluation of symptoms (Hamilton Rating Scale for Depression, Beck Depression Inventory, etc.) According to the Diagnostic and Statistical Manual, Version IV (DSM-IV), major depressive disorder often is used to describe PSD if 5 or more symptoms of depression are present for 2 or more weeks. Treatment of PSD also improves cognitive impairments or limitations imposed by stroke, e.g., orientation, memory, language and hand-eye coordination.

Both individual and group psychotherapy help the post-stroke patient regain emotional control and adjust to the loss of function and compromised self-image/self-esteem. The mode of therapy can be adapted to encompass any present cognitive and language deficits. Cognitive-behavioral, interpersonal, psychodynamic and eclectic forms of psychotherapy have been successful to alleviate or subdue many of the PSD symptoms. Inpatient and outpatient patient stroke groups, spousal support groups and discharge support groups are paramount to a smooth transition from hospital to rehab to aftercare venues, whether in long-term communal care or return to home.

Antidepressants

Antidepressants and psychostimulants often are the key medications used to help the post-stroke individual through recovery from depression. However, there is a huge caveat to consider: These medications may cause severe adverse effects in the elderly population increasing risk of falls, a primary confounder of safety in this population. Psychostimulants may be useful for reducing the negative effects of PSD, e.g., anhedonia, and promoting return of executive functioning, such as organization, goal setting and achievement, initiating activities and self-monitoring. A combination of methylphenidate and Prozac during the early stroke recovery period can dramatically improve mood, apathy and initiation by 50-82 percent.

Antidepressants from the classifications of tricyclics, SSRIs and newer atypical groups are used, according to the symptoms demonstrated by individual patients. Antidepressants are frequently prescribed for 1 year post-stroke; stopping medications before 1 year leads to vulnerability of developing another depressive episode.

Of course, individuation is always considered. When medication is indicated, most patients are advised to stay with a particular medication regimen for a minimum of 6 months, with reevaluation throughout the trial period. According to Living Well with Stroke, a randomized treatment efficacy nursing study for patients having PSD, there is no clear evidence of remission of depression in either the short- or long-run using SSRIs. Longer-term positive effects came with the addition of behavioral treatments that included cognitive restructuring and problem-solving/coping skills.

Tricyclics improve ADL function, improve appetite, lift general symptoms of depression and can help with insomnia, yet unfortunate anticholinergic side effects, especially in elderly stroke patients (dry mouth, dry eye, lowered GI motility, sedation, syncope, dysrhythmias, delirium) may cause discontinuation of this class of medication. SSRIs and the atypical antidepressants target serotonin and/or norepinephrine reuptake. Elevation of mood and improvement of depressive symptoms as rated on various depression rating scales frequently takes a minimum of 3-6 weeks and can take several months post-stroke. The addition of talk-based therapy during this time is highly encouraged.

Less traditional forms of assistance to lower the effects of PSD include music therapy, which tends to increase the production of dopamine to suppress aversive symptoms and pain; increases cross-hemispheric connectivity to music and lyrics; and enhances alertness, motivation, reward, attentiveness and executive functioning. Following middle cerebral artery stroke, brain plasticity and the laying down of renewed neural networks may be boosted. Music also has been demonstrated to modulate emotional arousal and decrease cortisol levels with respect to negative cardiovascular activity.

In summary, the goals and interventions for the nursing care of a patient with PSD include the following:

- Act quickly to encourage the patient or family members to report stroke and symptoms of depression.

- Educate the patient and family about the symptoms, prevalence and treatment of PSD.
- If depressive symptoms persist and interfere with the patient's ability to function, treatment should be initiated.
- Treat with medication and therapy for optimal relief from PSD.
- Obtain baseline and serial depressive symptom ratings throughout treatment.

Resources

Carod-Artal, F. (2010). Post-stroke depression: Can prediction help prevention? *Future Neurology*, 5(4), 569-580.

Lenzi, G., Alteri, M., & Maestrini, I. (2008). Post-stroke depression. *Rev Neurol*, 164(10), 837-40.

Mitchell, P., Veith, R., Becker, K., et al, (2008). Brief psychosocial-behavioral intervention with antidepressant reduces poststroke depression significantly more than usual care with antidepressant. *Stroke*, 39, 543.

Poynter, B., Shuman, M., Diaz-Granados, N., et al. (2009). Sex differences in the prevalence of post-stroke depression: A systematic review. *Psychosomatics*, 50(6), 563-569.

POST STROKE DEPRESSION ARTICLE QUESTIONS

1. Which of the following statements about post-stroke depression (PSD) is true?

- It is undertreated.
- It is limited to older patients.
- PSD is recognized as a problem by most caregivers.
- It occurs only in patients with left-sided strokes.

2. Dysthymic reactive depression in patients affected with a stroke:

- responds quickly to treatment with antidepressants
- includes less than 10 percent of all patients affected with this type of depression

- c. has no connection to the brain lesion locus
- d. rarely progresses to a major depressive disorder

3. Which of the following is NOT an indicator for reactional depression?

- a. aphasia
- b. dementia
- c. hopelessness
- d. amnesia

4. Women ages 45-54 have twice as many strokes as men because women:

- a. have more medical disabilities prestroke than men
- b. have less-effective coping skills than men
- c. have greater stress levels than men
- d. are non-compliant with preventive care measures

5. The antidepressant of choice for depression in post-stroke patients that features few cardiac side effects is:

- a. tricyclics
- b. selective serotonin reuptake inhibitors
- c. monoamine oxidase inhibitors
- d. serotonin-norepinephrine reuptake inhibitors

6. How long should antidepressants be initially prescribed for patients with strokes?

- a. less than 3 months
- b. 6 months
- c. 1 year
- d. until there is increased socialization in the patient

7. Cerebral ischemia in strokes, particularly in right-sided strokes, often results in:

- a. manic responses
- b. catastrophic responses
- c. suicidal ideation
- d. social decompensation

8. Anhedonia is:

- a. denial of disability
- b. poor speech comprehension
- c. severe lack of interest in people/interests
- d. short-term periods of amnesia

9. According to the DSM IV, major depressive disorder is used to describe PSD if:

- a. two symptoms are present for 1 month
- b. five or more symptoms persist for 2 or more weeks
- c. hopelessness and social withdrawal are present for 1 week
- d. two symptoms are present for 1 or more weeks, and one of these is confusion

10. Which of the following therapies is indicated for patients with strokes to stimulate the release of dopamine and modulate emotional response?

- a. cognitive-behavioral therapy
- b. movement therapy
- c. music therapy
- d. group therapy