Introduction to Learning Needs Packet Construction

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September 4, 2019
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PBL Session


Facts
Ideas/hypothesis
Learning Needs
Resource collection
Construct LN Package
Acquire and apply knowledge
Verna Williams
Part 1

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Ideas/hypothesis
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Part 1

What is a Learning Need Packet?

- Derived from the need to learn a topic to address the hypotheses/ideas and understand the patient problem
- Assigned to each student during the PBL session
- Constructed independently by each student
- Produced in form of a digital PDF or word document

File name

- Save all the learning needs files in the following format:

"PBL.case#.Part#.Group#.Initials.Title (topic)"

Example:

PBL.2301.Part1.Z3.MZD.Vital signs

Header

- Case number 2301
- Case title Verna Williams
- Part of case Part 1
- Title "Vital Signs"
- Name of student Maggie David
- Group number Z3

LN Package Components

Final document

1. Summary
2. Patient analysis
3. Hypothesis/Idea validation
4. Content
5. References

How to proceed

1. References
2. Content
3. Hypothesis/Idea validation
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How to proceed…..

1. References – sources
2. Content
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References / Sources

- Search for sources that will provide the information needed according to the assigned learning need:
  - Electronic or physical Books (reliable and up-to-date)
  - Text books (doctoral level)
  - Scientific research journals
  - Evidence-based sources
  - Reliable websites (government, universities, foundations, etc
  - Google Images (if verified reliable source of origin)

- List all resources used in the learning need (proper and complete citations)

- Add a note as to what information you got from what source.

Suggested sources

Useful Textbooks Available at the Wilson Dental Library compiled by the faculty:

Useful Textbooks Available at the Wilson Dental Library

Subject Category	Title	Author	Call #	E-book available?	
	Subject Guides also available at: http://wilson.usc.libguides.com/browse.php				
ANATOMY	Anatomical Basis of Dentistry	Liebgott, B.	QS 4 L716a 2011	Y	WDL Reserves
ANATOMY	Clinically Oriented Anatomy	Moore & Dalley	QS 4 M822c 2014	Y	WDL Reserves
ANATOMY	Human Anatomy & Physiology	Marieb, E.	QS 4 M334h 2016		WDL Reserves
ANATOMY	Principles of Anatomy and Physiology	Tortora & Derrickson	QS 4 T712p 2012		WDL Reserves
ANESTHESIA /SEDATION	Handbook of Local Anesthesia	Malamed, S.	WO 460 M236h 2013	Y (but 5th ed.)	WDL Reserves
ANESTHESIA /SEDATION	Handbook of Local Anesthesia Administration DVD (DVD)	Malamed, S.	mini WO 460 M236 2013		WDL Reserves
ANESTHESIA /SEDATION	Sedation: A Guide to Patient Management	Malamed, S.	WO 460 M236s 2010	Y	WDL Reserves
BIOCHEMISTRY	Lehninger Principles of Biochemistry: With an Extended Discussion of Oxygen-Binding Proteins	Nelson & Cox	QU 4 L524p 2008		WDL Reserves
BIOCHEMISTRY	Mark's Basic Medical Biochemistry: A Clinical Approach	Smith, et. al.	QU 4 S6439m 2013	Y	WDL Reserves
BONE BIOLOGY	Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism, 7th	Ed: Cliff Rosen	WE 250 P953 2013	Y	WDL Reserves
BONE BIOLOGY	Principles of Bone Biology	Bilezikian, et. al.	WE 200 P957 2008		WDL Reserves
DENTAL ANATOMY	Anatomy of Orafacial Structures	Brand, I.	WU 101 B817a 2014		WDL Reserves
DENTAL and ORAL HISTOLOGY	Ten Cate's Oral Histology: Development, Structure and Function	Nanci, A.	WU 101 T289o 2013		WDL Reserves
DENTAL ANATOMY	Wheeler's Dental Anatomy, Physiology, and Occlusion	Ash & Nelson	WU 101 W564t 2015	Y (but 9th ed.)	WDL Reserves

And more……..

https://libraries.usc.edu/

Class of 2023 Intranet

References / Sources

- If you have trouble finding the information you need……

ASK FOR HELP!

- Librarians
- Facilitator
- Group members
- Faculty

Examples of Citation

Book:

Kumar V, Cotran RS, Robbins SL. Robbins Basic Pathology. 7th ed., W.B. and Suanders Company; 2007: Chapter 11, 13. Print.

e-Book:

Taylor Palmer, "Chapter 9. Agents Acting at the Neuromuscular Junction and Autonomic Ganglia" (Chapter). Brunton LL, Lazo JS, Parker KL: **Goodman & Gilman's The Pharmacological Basis of Therapeutics**, 11th Edition: http://www.accessmedicine.com/content.aspx?aID=957196

Journal Article:

Rothwell PM. Algra A. Amarenco P. Lancet. 377(9778):1681-92, 2011 May 14. [Journal Article. Review].

https://www.ncbi.nlm.nih.gov/pubmed/21571151

Website:

http://www.cda.org/Portals/0/pdfs/fact_sheets/gum_disease_english.pdf

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Content

- Serves as main source of information to acquire or advance knowledge on your topic
- Information pertinent to testing the validity of the hypothesis/ideas assigned to the student
- Start with a general overview and then focus on the particular

- Example:
  - Learning need : Aspirin
  - Content:
    - What type of drugs does aspirin belongs to?
    - Commercial and generic names
    - Structure
    - Mechanism of action (illustrate)
    - Pharmacokinetics
    - Doses
    - Uses
    - Contraindications
    - Side effects
    - Drug interactions

Where am I going to get this information: Pharmacology textbooks, Pharmacological databases

Content

- Information extracted from dependable sources. If possible, always start with a text book and update/complement from other sources. However, if the same, you don't need to repeat information from the other sources. Edit to avoid repetition.
- You can cut and paste into a working file (WORD™ or Google drive file)
- Appropriate level of knowledge. Do not use books, magazines or websites designed to patients. You are the doctor not the patient.
- Well organized, include as many pictures, diagrams, flow charts, tables, etc. If you are thinking about it, illustrate!
- Use several different sources for your content, Make it easy to read: create paragraphs, use the same font (not too small not to big), embed the pictures or tables in the text, highlight, etc. It makes a big difference!

Content

The tight junctions between capillary endothelial cells in the brain and between the epithelial cells in the choroid plexus (a highly vascular portion of the pia mater that projects into the ventricles of the brain and is thought to secrete the cerebrospinal fluid) effectively prevent proteins from entering the brain in adults and slow the penetration of smaller molecules. An example is the slow penetration of urea (Figure 32–5). This uniquely limited exchange of substances into the brain is referred to as the blood–brain barrier. Some physiologists use this term to refer to the barrier in the capillary walls and the term blood–CSF barrier to refer to the barrier in the choroid epithelium. However, the barriers are similar, and it seems more appropriate to use the term blood–brain barrier to refer to exchange across both barriers.

Passive diffusion across the tight cerebral capillaries is very limited, and little vesicular transport takes place. However, there are numerous carrier-mediated and active transport systems in the cerebral capillaries. These move substances out of as well as into the brain, though movement out of the brain is generally freer than movement into it because of bulk flow of CSF into venous blood via the arachnoid villi.

II. Penetration of Substances into the Brain:

Water, CO2, and O2 penetrate the brain with ease. So do the lipid-soluble free forms of steroid hormones, whereas their protein-bound forms and, in general, all proteins and polypeptides do not. The easy penetration of CO2 contrasts with the slow penetration of H+ and HCO3– and has physiologic significance in the regulation of respiration.

Glucose is the major ultimate source of energy for nerve cells. Its passive penetration of the blood–brain barrier is slow, but it is transported across the walls of brain capillaries by the glucose transporter GLUT 1. The brain contains two forms of GLUT 1: GLUT 1 55K and GLUT 1 45K. Both are encoded by the same gene, but they differ in the extent to which they are glycosylated. GLUT 1 55K is present in high concentration in brain capillaries (Figure 32–6). Infants with congenital GLUT 1 deficiency develop low CSF glucose concentrations in the presence of normal plasma glucose, and they have seizures and delayed development.

Another transporter in the cerebral capillaries is a unique Na+–K+–2Cl– cotransporter that is stimulated by ET-1 and ET-3 and apparently induced by a humoral factor from astrocytes. It may help keep the brain K+ concentration low. In addition, transporters for thyroid hormones, several organic acids, choline, nucleic acid precursors, and neutral, basic, and acidic amino acids are present.

A variety of drugs and peptides actually cross the cerebral capillaries but are promptly transported back into the blood by a multidrug nonspecific transporter in the apical membranes of the endothelial cells. This P-glycoprotein is a member of the family of ATP-binding cassettes that transport various proteins and lipids across cell membranes. In mice in which the function of this cassette has been disrupted by gene inactivation, much larger proportions of systemically administered doses of various chemotherapeutic drugs, analgesics, and opioid peptides are found in the brain than in controls. If pharmacologic agents that inhibit this transporter can be developed, they could be of value in the treatment of brain tumors and other CNS diseases in which it is difficult to introduce adequate amounts of therapeutic agents into the brain.

III. Function of the Blood–Brain Barrier:

The blood–brain barrier probably maintains the constancy of the environment of the neurons in the central nervous system. These neurons are so dependent on the concentrations of K+, Ca2+, Mg2+, H+, and other ions in the fluid bathing them that even minor variations have far-reaching consequences. The constancy of the composition of the ECF in all parts of the body is maintained by multiple homeostatic mechanisms, but because of the sensitivity of the cortical neurons to ionic change, it is not surprising that an additional defense has evolved to protect them.

Other suggested functions for the blood–brain barrier is protection of the brain from endogenous and exogenous toxins in the blood and prevention of the escape of neurotransmitters into the general circulation.

IV. Development of the Blood–Brain Barrier:

In experimental animals, many small molecules penetrate the brain more readily during the fetal and neonatal period than they do in the adult. On this basis, it is often stated that the blood–brain barrier is immature at birth. Humans are more mature at birth than rats and various other experimental animals, and detailed data on passive permeability of the human blood–brain barrier are not available. However, in severely jaundiced infants with high plasma levels of free bilirubin and an immature hepatic bilirubin-conjugating system, free bilirubin enters the brain and, in the presence of asphyxia, damages the basal ganglia (kernicterus). The counterpart of this situation in later life is the Crigler-Najjar syndrome in which there is a congenital deficiency of glucuronyl transferase. These individuals can have very high free bilirubin levels in the blood and develop encephalopathy. In other conditions, free bilirubin levels are generally not high enough to produce brain damage.

V. Clinical Implications:

Physicians must know the degree to which drugs penetrate the brain in order to treat diseases of the nervous system intelligently. For example, it is clinically relevant that the amines dopamine and serotonin penetrate brain tissue to a very limited degree but their corresponding acid precursors, L-dopa and 5-hydroxytryptophan, respectively, enter with relative ease.

Another important clinical consideration is the fact that the blood–brain barrier tends to break down in areas of infection or injury. Tumors develop new blood vessels, and the capillaries that are formed lack contact with normal astrocytes. Therefore, there are no tight junctions, and the vessels may even be fenestrated. The lack of a barrier helps in identifying the location of tumors; substances such as radioactive iodine-labeled albumin penetrate normal brain tissue very slowly, but they enter tumor tissue, making the tumor stand out as an island of radioactivity in the surrounding normal brain. The blood–brain barrier can also be temporarily disrupted by sudden marked increases in blood pressure or by intravenous injection of hypertonic fluids.

Breakdown of the BBB

Breakdown of the blood–brain barrier (BBB) is an important contributing factor to injury in many brain diseases, including stroke. A number of different, partially independent components, including the extracellular matrix, tight junctions (TJs), pericytes, and astrocyte endfeet, together with adherens junctions, form junctional complexes and play a central role in the control of BBB permeability and maintenance of cell polarity (Bazzoni et al., 2000; Zlokovic, 2008). After stroke in the adult, BBB disruption can either occur transiently, in two distinct phases (Belayev et al., 1996; Rosenberg et al., 1998), or be continuous (McColl et al., 2008) during the acute injury phase, with the extent and timing dependent on age, genetic background, and gender. Stroke severity is also exacerbated by predisposing factors, such as infection or systemic inflammation, that affect various components of the BBB (Denes et al., 2010).

Once the blood-brain barrier is broken down, it can harm our body in many ways. For example, in some patients, Alzheimer's disease may be caused (or more likely, aggravated) by a breakdown in the blood–brain barrier[3].

Based on researches, it's known that the BBB can be weaken by[3,7]

Meningeal Invasion

There are three potential bacterial routes of entry into the CSF: hematogenous, via a contiguous structure, and direct implantation. The hematogenous route is the most common; the primary foci of infection may be the nasopharynx, skin, lung, heart, gastrointestinal or genitourinary tract, umbilical stump, or elsewhere. Bacteria may enter the CSF through the dural sinuses or the choroid plexuses,the precise mode of penetration is unknown. There

Content

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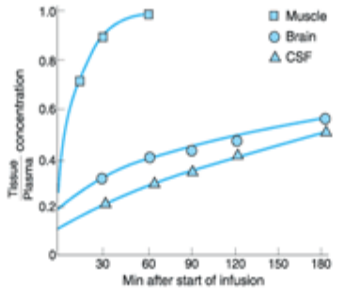

Figure 32-5. Penetration of urea into muscle, brain, spinal cord, and CSF. Urea was administered by constant infusion.

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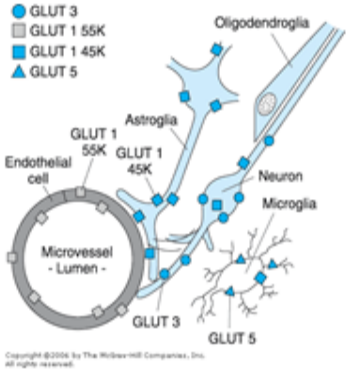

Figure 32-6. Localization of the various GLUT transporters in the brain.

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How to proceed…..

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3. Hypothesis/Idea validation
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Hypothesis/Idea Validation

- State and analyze each one of the ideas assigned to your learning need
- Validate or invalidate the idea based on your learning need
- Provide an explanation as why you validate/invalidate the idea
- Revise the idea based on your newly acquired knowledge

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Summary

sum·ma·ry

noun

1. a brief statement or account of the main points of something.

"a summary of Chapter Three"

synonyms: synopsis, précis, résumé, abstract, digest, encapsulation, abbreviated version

adjective

1. dispensing with needless details or formalities; brief.

"summary financial statements"

synonyms: abridged, abbreviated, shortened, condensed, concise, capsule, succinct, short, brief, pithy; formal compendious "a summary financial statement"

What is in Summary?

- A brief description of the major points or highlights of your content
- Not a repetition of the facts
- No more than one page
- Do not place tables, pictures, diagrams, etc
- Enough information and detail to understand all aspects of the topic
- Not designed to replace the content

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Patient Analysis

Apply the information you learned to the case

- Search, copy, paste, read, understand and then: apply!
- How this knowledge helps you understand what is going on with the case
- Explain with your own words what is happening to the patient based on what you learn
- Do not repeat the facts! Only what is necessary for your analysis.

Example

Learning need: Hepatic panel lab values

Patient analysis: how are the patient's values? If they are normal, what does it mean? If they are abnormal, what does it mean? Why where those tests done? What are the consequences of those values? How it helps you understand the patient? What do you think is wrong with the patient?

Components

- Summary
- Patient analysis
- Hypothesis/Idea validation
- Content
- List of references

Grammar & Format

- Learning need packet should be free of grammatical and spelling errors
- Edit to avoid repetition
- Appropriate length: There are NO minimum pages! There are NO maximum pages! Will always depend on the topic!
- Should have all the information necessary to acquire the appropriate knowledge of the topic and address the hypothesis/ideas
- Don't skip parts that you think you know….maybe other members of the group don't!

Last advice

- Send/upload your learning needs packets on time
- Read all learning needs and come prepared to the sessions.