

Learning a new topic can be difficult!

Sometimes students don't know what is going to be covered in class (it is a "free flow" of information). This "free flow" can become a jumble or be confusing.

Even if students know what they will cover in class, they often do not know what information they are supposed to take away from that material. This means they don't know "what to listen to" most intently. They also don't know how their knowledge will be assessed.

Here are a few tips on how to solve this.

1. Provide a "class outline". This will give students an overview of what they'll cover in class. This will help students create a mental map of the subject. This will help, somewhat.
2. Provide "learning outcomes/objectives". This will tell students what they are supposed to take away from class material, and how their knowledge will be assessed. This will help a bit more.
3. Combine the two (class outline and course outcomes/objectives). This will likely help the most.
4. Use course outcomes to create good exam questions!
5. And... you can even go overboard and use learning outcomes to create self-assessment questions.

A few examples follow.

1. Class outline

The “class outline” lists what the instructor is going to cover. It is helpful because students get an idea of what will be discussed, and in what order (it creates a “mental map” of the subject). However, the outline does not specify what the students are supposed to “do” with that information, nor what they are supposed to “know” for each topic. Also, the outline does not tell the instructor how to judge if one has learned the material.

Here are a few examples.

Hypertension and antihypertensives

Outline

Definitions

Pathology

Physiology of blood pressure regulation

Treatment

Non pharmacological

Pharmacological

Sympatholytics

Calcium channel blockers

ACE Inhibitors & Angiotensin II antagonists

Vasodilators

Diuretics

Data management

Outline

Data definitions/types

Data record keeping/notebooks

Data protection/confidentiality

Data ownership, data stewardship

Data sharing/availability

Data management plans

2. Learning objectives/outcomes

There are different definitions of learning “objectives” and “outcomes”. I won’t go into them and will just call them “objectives/outcomes” here.

Learning objectives/outcomes help instructors and students understand what they are to “do” with the material they learned and how to tell if students learned it. They are specific and measurable and attainable (or even “SMART”: Specific, Measurable, Attainable, Relevant, and Time-bound).

They are usually something like “by the end of this lecture the students should be able to...” (then you list what they should be able to do).

Another advantage of course objectives/outcomes is that knowing them in advance, will help students “tune-in” to the relevant information (it has been shown that if you are alerted to “what you should know”, you will listen to that information with more focus, than if you don’t know what message you are supposed to take out of it).

Finally, good outcomes will help create good exam questions (point 4) and study-guides (point 5).

I found these sites useful when creating course outcomes:

<https://resources.depaul.edu/teaching-commons/teaching-guides/course-design/Pages/course-objectives-learning-outcomes.aspx>

<https://provost.utexas.edu/the-office/academic-affairs/developing-learning-outcomes/>

And this one useful when choosing verbs (list, describe, etc...):

<https://teaching.utoronto.ca/teaching-support/course-design/developing-learning-outcomes/appendix-b-useful-verbs-for-developing-learning-outcomes/>

I use 4-7 learning outcomes per 50 min of lecture. Here are a few examples.

Dementia

Learning objectives/outcomes

- List the major types of dementia and the approximate frequency of each
- List the treatable causes of dementia
- Recognize the clinical features of Alzheimer’s disease
- Describe changes in the brain in Alzheimer’s disease, and the role of APP, A β peptide, presenilins, and apolipoprotein E
- List genetic mutations that are associated with Alzheimer’s disease

Neurological and psychiatric disorders

Learning outcomes/objectives

Compare and contrast the disorders of seizures, dementia, Parkinson’s disease, schizophrenia, addiction, anxiety and mood disorders with respect to their:

- Clinical manifestations
- Level of heritability
- Etiology at a cellular, brain structure, neurotransmitter, or circuit level
- Main targets for treatment strategies

3. Pairing class outline with learning objectives/outcomes

I find it most useful to pair the outline with the learning objectives/outcomes in the same slide.

Here are some examples

Animal models of addiction	
Outline	Objectives/Outcomes
Value of human/animal studies General statistics Risk factors for addiction Strengths & limits of human studies	→ Explain advantages/disadvantages of using humans/animals when studying addiction
Simple animal models of addiction Self-administration Sensitization Conditioned place preference Drug discrimination	→ Describe and compare different animal models of addiction
Translational animal models of addiction DSM 5 criteria for addiction Self-administration	→ Compare and contrast animal models with human addiction
Critical concepts in experimental design	→ Explain critical concepts in experimental design, for the correct interpretation of results

Motives for drug taking	
Outline	Objectives/Outcomes
Operant conditioning ("behaviorism") Basic principles Application to "addiction"	→ Explain principles of operant conditioning
Issues with "behaviorism" In general With respect to addiction	→ Illustrate the paradoxical effects of rewards & punishments → Discuss how "lack of skills" reduces the effectiveness of behaviorism
Motives (initial reasons) for drug taking Enhancement: internal Enhancement: external Relief: internal Relief: external	→ Differentiate between motives of drug taking
Other reasons that lead to "addiction" Repeated exposure (medical, accidental)	→ Discuss pros/cons of stratifying people according to reasons for drug taking

Data management	
Outline	
Data definitions/types Data record keeping/notebooks Data protection/confidentiality Data ownership, data stewardship Data sharing/availability Data management plans	
Objectives/Outcomes	
<ul style="list-style-type: none"> Describe "data" (definitions/types) Describe core issues about data management and record keeping (integrity, confidentiality, availability) Describe the importance, and ethical/compliance issues relating to data accuracy (integrity) data protection (confidentiality), ownership and stewardship, and data sharing (availability) Discuss the importance of data management plans 	

4. Use course objectives/outcomes to create good exam questions

Learning outcomes/objectives can be used to create good exam questions, which match the course outcomes. Here are a few examples

Outcome 1

Explain mechanisms of neuromodulation

Outcome 1 Question (medium difficulty)

Describe ways in which neuromodulation can occur at the level of the cell body, the synapse, and post-synaptically.

Outcome 2

Provide different ways of classifying neurotransmission in CNS

Outcome 2 Question (easy)

There are different ways of classifying neurotransmission. Provide one classification. Include ALL the following:

Acetylcholine	Dopamine	Norepinephrine	Serotonin
GABA	Glycine	Aspartic acid	Glutamate
Nitric oxide	Neuropeptide Y	Neurotensin	

Outcome 3

List at least 4 families of neuropeptides, their precursors and active peptides

Outcome Question 3 (easy)

List at least 2 family of neuropeptides, their precursors, and active peptides EXCEPT do not include the opioid family nor the CRH family.

Outcome 4

Compare and contrast classical neurotransmitters & neuropeptides when it comes to: Size, Synthesis, Storage, Release, Degradation, and Speed of action. use this information to predict effects of co-released neurotransmitters & neuropeptides of known function

Outcome 4 Question (medium difficulty)

"Classical" neurotransmitters (especially amino acid neurotransmitters) differ in many ways from neuropeptides. Fill the table below to describe main ways they differ when it comes to size, synthesis, storage, release, degradation, and speed of action

	Neurotransmitters	Neuropeptides
Size		
Synthesis		
Storage		
Release		
Degradation		
Speed of action		

Outcome 4 Question (medium difficulty)

Imagine that the same neurons of the central nucleus of the amygdala release both GABA (an inhibitory “classical” neurotransmitter that DECREASES ANXIETY) and CRH (a neuropeptide that INCREASES ANXIETY). What happens to anxiety when you stimulate these neurons of the amygdala? Hint: think about how neuropeptides are released in a “frequency-dependent” manner.

Outcome 5

Describe main properties and mechanisms of action of opioid peptides and CRH functioning as a neuropeptide and utilize this information to design experiments or predict outcomes testing their function

Outcome 5 Question (Difficult)

Opioid peptides bind to mu opioid receptors in the midbrain (the ventral tegmental area, VTA), and indirectly increase the activity of dopamine neurons. Describe the mechanism by which this increase occurs (you may provide a drawing to complement your description).

Outcome 5 Question (medium difficulty)

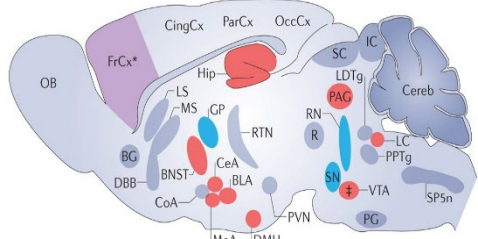
Opioid neuropeptides come from different precursors, they act on different receptors (R), and they have different effects. Complete the table below to provide this information

Precursor	Neuropeptide	R	Main effects
Pro-opioid melanocortin (POMC)			
Prepro-enkephalin			
Prepro-dynorphin			

Outcome 6 Question (easy)

This figure is taken from a review by Hanckens et al., titled “Region-specific roles of the corticotropin-releasing factor–urocortin system in stress” (Nat Rev Neurosci, 2016).

a Effect of CRFR1 activation on anxiety-like behaviour



Legend: CRFR2: corticotropin releasing factor receptor type 2. BG, basal ganglia; BLA, basolateral amygdala; BNST, bed nucleus of the stria terminalis; CeA, central amygdala; Cereb, cerebellum; CingCx, cingulate cortex; CoA, cortical amygdala; DBB, diagonal band of Broca; DMH, dorsomedial hypothalamus; FrCx, frontal cortex; GP, globus pallidus; Hip, hippocampus; IC, inferior colliculus; LC, locus coeruleus; LDTg, laterodorsal tegmental nucleus; LS, lateral septum; MeA, medial amygdala; MS, medial septum; NTS, nucleus of the solitary tract; OB, olfactory bulb; OccCx, occipital cortex; PAG, periaqueductal grey; ParCx, parietal cortex; PG, pontine grey; PPTg, pedunculopontine tegmental nucleus; PVN, paraventricular nucleus of the hypothalamus; R, red nucleus; RN, raphe nucleus; RTN, reticular nucleus; SC, superior colliculus; SN, substantia nigra; SON, supraoptic nucleus; SP5n, spinal trigeminus nucleus; VMH, ventromedial hypothalamus; VTA, ventral tegmental area.

Based on this figure, what do you think will happen if you micro-infuse corticotropin releasing factor (CRF) in the bed nucleus of the stria terminalis (BNST)? (one sentence is sufficient)

What about in the basolateral amygdala (BLA)? (one sentence is sufficient)

What about in the globus pallidus (GP)? (one sentence is sufficient)

5. Going a bit overboard: using learning outcomes/objectives to self-assess

Learning outcomes/objectives can be used to create questions for students to self-assess. I did this for a course I taught with over 120 students (PHM 480C, eCIS score of 4.8). Students appreciated this very much, although it is somewhat “spoon-feeding” and more appropriate to undergraduate vs. graduate students.

Here are some examples of learning outcomes/objectives with SOME questions (not exhaustive).

I pre-empted it with: “Students: don’t just say “yes, I can do this”, actually give it a try!”

Seizures learning outcomes/objectives

- *Define seizure, epileptogenesis, epilepsy, and status epilepticus.*

Can you define those terms?

- *Explain how seizures develop (i.e. epileptogenesis)*
- *List possible precipitating factors of seizures*

Which ion channels are MOST involved at the single-cell level?

How about at the circuit level?

Can you describe the interplay between GABA and Glutamate during a tonic-clonic seizure?

- *Differentiate the types of seizures and describe their clinical presentation*

If a seizure is described (e.g. blank stare, movement of hand only, movement of hand then generalized, start with generalized, drop suddenly, small jerky movements etc...) Can you name what type of seizure it was? Remember to distinguish “impaired of consciousness” with no impaired of consciousness

Conversely, if you see the name of a type of seizure, can you describe its symptoms?

At what ages does the disorder manifest itself?

- *List disorders that lead to secondary epilepsy*

Can you list these disorders? (they are listed in the handout)

- *List genetic mutations that have been associated with epilepsies*

Can you list or at least recognize the genetic mutations that have been associated with epilepsies?

Dementia learning outcomes/objectives

- *List the major types of dementia and the approximate frequency of each*

Can you list them and do you know the approximate frequency of occurrence of each?

- *Explain the treatable causes of dementia*

Can you list and explain the treatable causes of dementia? E.g., is it easier to treat someone with Alzheimer’s disease or with an insufficiency of vitamins B?

Is nicotine (and acetylcholine receptor activation) good or bad for treating dementia?

- ***Describe the neurocognitive domains, and how they are affected by dementia***

Can you describe all 5 of them, and which ones are affected by dementia?

Can you tell which ones match with which brain region?

Can you match neurocognitive domains to brain regions? (e.g. practice exam question-like)

- ***Recognize the clinical features of Alzheimer's disease***

Can you describe a patient with Alzheimer's disease, and how the disease progresses?

At what age does it manifest itself and why?

- ***Describe changes in the brain in Alzheimer's disease, and the role of APP, A β peptide, presenilins, and apolipoprotein E***

Can you describe what happens with all of those "players"?

What does accumulation of A β peptide lead to? (EXTRAcellular amyloid neuritic plaques)

What does hyperphosphorylation of Tau lead to? (INTRAcellular neurofibrillary tangles, due to destabilization of microtubules)

Apolipoprotein E: might facilitate "clumping" of A β peptides.

- ***List genetic mutations that are associated with Alzheimer's disease***

Can you list them?

Can you list which type of ApoE is protective (E2) vs risk-factor (E4) vs most common (E3)?