

Review Questions for Midterm #1 + Answers

What follows below are answers to all of the review questions that are likely to be part of the material for Midterm #1, which will take place on Thursday, October 19, during our regular time. Remember that the midterm will be held in several locations: please consult the website if you're not clear about where to go.

I have supplied some reasonable answers for each of the review questions below. If you have any questions, just let me know.

Remember that * = those questions that you do not need to know (these are questions that I have gone over in previous years, but not this year).

1. * Describe the model of the individual in psychology (See Lecture Outline #1).

You don't have to worry about this.

2. What is the hindsight bias and how was it demonstrated in the Psychology Pre-Test demonstration? (see Myers, Chapter 1 for details)

Recall that in the Psychology Pre-Test, most of the class performed below chance levels (that is, below what Koko the gorilla would be expected to perform). Why is this? The Pre-Test demonstrates that psychology really isn't a study of the obvious (otherwise, as a group, the class would perform much above chance levels). Why then do we think that psychology is the study of the obvious? Because when we hear of some research finding in psychology, our evaluation of whether that finding makes sense tends to be influenced by our tendency to retrieve information from memory that is consistent rather than inconsistent with that finding.

3. What does it mean to say that behavior is multidetermined?

This is a basic principle of psychology: for every given behaviour, there are multiple causes. As an example, consider the causes for a child aggressing in a particular situation. There are multiple causes—could be that his parents were aggressive to him when he was young, and/or he was

4. What is determinism?

There were two assumptions of psychology (and science); this is the first one. Determinism is the assumption that all effects have causes (or put in a slightly different way, there is order in the universe). And thus, the goal of science is to discover/uncover those causes.

5. * What is the relationship between determinism and free will?

There are different meanings for determinism. And one philosophical/theological use of the term determinism is to distinguish it from free will. In this use of determinism, philosophers mean that one's actions are pre-determined. The opposite of this kind of determinism is free will.

6. What is the relationship between belief in determinism and political philosophy?

There is a tendency for politically conservative people to believe that causes of behavior reside with the individual. Thus, they are less inclined to support social welfare/assistance programs and other external, environmental factors. In contrast, politically liberal people believe that the causes of behavior should also include those external, environmental factors, and thus, they are more likely to support social welfare/assistance programs.

7. * What kind of determinism do scientists utilize in their work?

A moderate form of determinism: they simply acknowledge that all effects have causes and set out to discover those causes.

8. What is empiricism? How do scientists use empiricism in their work?

Empiricism was the second assumption of psychology/science. It provides a set of guidelines for what kinds of information/knowledge are acceptable/amenable/admissible for scientific study

9. What is empirical knowledge?

Knowledge gained through (at least one of) the five senses.

10. What is the one important criterion of any scientific theory or hypothesis? Why is it so important?

A scientific theory must be capable of disconfirmation: it must be possible to disprove the theory through empirical research. (The philosopher, Karl Popper, once said that, "a theory that can never be killed can never be truly alive.") A theory is no good if you can't critically test it in research.

11. What is an operational definition? What are the two criteria that scientists use to evaluate operational definitions?

An operational definition is the same as a measure of a construct. This sounds abstract, so let's explain with an example: suppose you were interested in studying intelligence, and you gave people an IQ test. In this example, intelligence = the construct and IQ test = a measure of that construct. It is important to distinguish between a construct and a measure. OK--so how good is a measure (operational definition) of a construct? There are two criteria: validity and reliability...

12. Know the distinction between validity and reliability.

Validity refers to whether a measure/operational definition is accurate, truthful. One way to evaluate a measure for its validity is to ask the question, "Does this measure capture the construct--is this measure prototypic of the construct?" OK--concrete example: in our discussion of measures/operational definitions of aggression, we agreed that physical aggression--harming another person physically--was a very valid measure of aggression because physical aggression captures what we mean when we think about aggression. In contrast, a measure of aggression like yelling would have lower validity because: (1) it's not really a prototypic example of aggression, and (2) yelling could be a measure of other constructs that we aren't interested in when we're studying aggression (e.g., excitement)--in other words, yelling would have LOWER discriminant validity because it could be the result of other constructs besides aggression. It's bad to use a particular measure when that measure could be confused with other constructs.

In contrast, reliability refers to how stable the data are. In my example of two observers of a classroom who rate the students on their aggression. If the two observers agree substantially on their ratings of aggression in the classroom, then this measure (aggression ratings) has high reliability; if they don't agree very much, then this measure has low reliability. Low reliability is bad because it says that you can't trust the numbers very much--that they are unstable, dependent on who is doing the ratings, etc.

Validity is associated with the word "accuracy"; and reliability is associated with the word "precision."

13. How does the story of Broca's brains (he used brain size as a measure of intelligence) illustrate the difference between validity and reliability?

Broca was a careful scientist, so his data were highly reliable--the measurement of brain size was very reliable--two different people weighing the same brain would get nearly exact values. But brain size has zero validity as a measure of intelligence. Thus in Broca's research we have a very reliable measure but it has no validity.

14. Contrast correlational research with experimental research: what are the benefits and pitfalls of both?

I didn't really go into this question except to say that experimental research can establish causes and correlational research cannot. But please note the material at the bottom of the handout on the two experiments on TV watching and aggression: experiments can demonstrate that X causes Y, It doesn't tell you that X is the only cause of Y (because, of course, behavior is multidetermined). It also can't determine that X is the strongest (or most important) cause of Y.

15. What feature of a good survey allows us to generalize the results of the survey to the population?

The survey must have a sample that is representative of the population--you must have a representative sample.

16. What is a representative sample? Why is it important in survey research?

A representative sample is one whose characteristics match those of the population. For example, if the population of Canadians consists of 52% women and 48% men, if your sample has around 52% women, the sample is representative of the population with respect to gender. If your sample has, say, 70% women, then your sample is not representative of the population with respect to gender. If 25% of your sample is over the age of 75, and so is the population of Canada as a whole, then your sample is representative with respect to citizens over 75.

Having a representative sample is critical because having one allows you to take the results of your sample and apply/generalize them to the population. So if your survey of only 1000 Canadians in which you find that 65% of your sample supports the Liberal party is a representative sample of Canadians, that means you can now say that 65% of all Canadians supports the Liberal party (with some sampling error).

What's the best method for obtaining a representative sample? Employ random sampling. What's random sampling? Basically--in random sampling, everyone in the population has an equal chance of being chosen for the sample.

17. How did the Literary Digest poll of 1936 demonstrate the importance of random sampling (obviously, you don't have to know the numbers--just the principle!)

As you know, Literary Digest was a literary magazine in the U.S. that had conducted an election poll during the U.S. presidential elections for the 5 elections before 1936. They had correctly predicted the winner in each of those prior 5 elections.

In 1936, they conducted the largest poll/survey ever: they sent out 10 million surveys and got 2.4 million back. Out of the 2.4 million respondents, 57% Landon and 43% Roosevelt. But the actual election, 38% Landon and 62% Roosevelt. What happened? Two sources of potential bias in their sample:

1. Sampling bias: LD sent surveys to people listed in phone books, club memberships, etc. This made the sample biased in the direction of wealthier people.
2. Nonresponse bias: only 24% of people responded to the survey. There are often differences between those who respond to a poll and those who don't. This might possibly have biased the survey even more.

These two sources of bias led to a sample that was unrepresentative of the population. If they had used random sampling, the poll would have been closer to representative.

18. How did the Literary Digest poll of 1936 demonstrate problems with biased samples?

See #17

19. What were the biases that led to the problems with the Literary Digest poll of 1936?

See #17

20. How does the Literary Digest poll of 1936 illustrate the difference between reliability and validity (hint: sampling error is related to reliability and accuracy is related to validity)?

Validity = accuracy and reliability = precision. Now the LD poll was very precise because 2.4 million respondents leads to an estimate of the population that has very little sampling error (from the handout on sampling error, $N = 2.4$ million leads to a sampling error of $\pm 0.06\%$). But because of sampling bias, this very precise estimate was completely inaccurate. In short, the LD poll had extremely high reliability but really low validity.

21. What is sampling error?

See the handout on sampling error from class. Remember that any sample will differ from the population value: thus, even though when we flip a coin 10 times, we expect 5 heads and 5 tails, any sample of 10 coin flips won't always give you exactly 5 heads and 5 tails. Sometimes we get 6 heads and 4 tails, sometimes 3 heads and 7 tails. Thus, each sample is associated with "error."

22. What is the law of large numbers? (You don't have to know the numbers--just the principle!)

Statistical principle: under random sampling, the only factor that determines sampling error is the sample size, N . (Specifically--as N goes up, sampling error goes down.)

23. What is the relationship between sample size of a survey and sampling error (and precision)? What is the relationship between proportion of the population sampled and sampling error/precision (i.e., the sample-to-population ratio)?

N is the only factor that determines sampling error; the sample-to-population ratio is essentially unimportant. (Remember: this is the "counterintuitive" principle that I mentioned in class.)

24. What does it mean for two variables to be positively correlated? Negatively correlated? Uncorrelated?

Positive correlation: as one variable goes up, the other also goes up (e.g., between hours studying for a midterm and one's performance)

Negative correlation: as one variable goes up, the other goes down (e.g., between number of beers consumed before a midterm and one's performance on that midterm)

Zero correlation: as one variable goes up, the other neither goes up nor goes down on average (e.g., between height and one's performance on a midterm)

25. What are the three causal possibilities when there is a correlation between two variables?

$X \rightarrow Y$, $Y \rightarrow X$ (reverse causality), $Z \rightarrow$ both X and Y (spuriousness). I've gone over this many times in my lectures.

26. What is a spurious correlation? What does the "third variable" refer to?

Spurious correlation: X and Y are correlated not because there is any causal relationship between them but because they are both caused by a third variable, Z . Example: crime rate and ice cream sales.

27. Know how to identify the parts of an experiment: conditions, independent variable, dependent variable.

Example: Bandura et al.: kids were randomly assigned to two conditions: aggressive model vs. nonaggressive model. Thus the independent variable that was manipulated was aggressiveness of model. Dependent variable was the outcome: how the kids behaved in the presence of their own Bobo doll.

28. Why is random assignment to conditions so important in an experiment? What does it eliminate?

Random assignment eliminates all possible sources of confounding (e.g., alternative explanations) except for the independent variable that was manipulated in the experiment. Even though behavior is indeed multidetermined, those other causes of behavior are controlled for when you randomly assign people to conditions. So—in the Bandura et al. study, the difference between the two conditions could not be explained by “all of the naturally aggressive kids were in the aggressive model condition” because random assignment equated the number of naturally aggressive kids in the two conditions. And remember that nothing that is the same between two conditions can explain the differences between the two conditions.

Random assignment doesn't eliminate the multiple causes of behavior. It just makes it impossible for those other causes to explain the differences between the experimental conditions. The only thing that can explain the differences between the experimental conditions in the dependent variable is whatever differed in the independent variable.

29. Know the distinction between random sampling (in survey research) and random assignment (in experimental research)

Should be clear from my explanations of the two concepts in previous questions.

30. What are the benefits of experimental research? What does it not tell us?

Experiments can tell you that X causes Y. Even though there are many causes of Y, a well-designed experiment can demonstrate that X is one of those causes.

Experiments cannot tell you that X is the only cause of Y; they cannot tell you that X is the most important or strongest cause of Y.

31. Define external validity and internal validity. Define discriminant validity.

External validity: the extent to which the experimental situation corresponds to some situation in the real world / the extent to which you can generalize the results of the experiment to some situation in the real world.

Internal validity: the extent to which your experiment can say that it was really X that caused the differences between conditions rather than some other factor.

Discriminant validity: related to internal validity—allows you to say that your measure of X really is a measure of X and not some other construct that we don't want to study. Example: when I went over possible measures of aggression. We agreed that physical aggression (e.g., number of times one kid hits another kid) was a very good measure of the construct of aggression because there were few, if any, other reasons for hitting besides aggression—in short, that measure would have high discriminant validity. In contrast, consider yelling as a measure of aggression: although yelling might be associated with aggression sometimes, there are other constructs that could also lead to yelling—excitement, naturally loud voice, and we wouldn't want yelling to be our measure of aggression because of these alternative explanations for yelling besides aggression. Thus, yelling would have low discriminant validity. If we used yelling as a measure of aggression, we couldn't be sure that yelling didn't occur because of those non-aggressive sources. In short, we want measures that are only related to the construct we care about (in this case, aggression) and not other confounding constructs (e.g., excitement).

32. What is reliability? Why do we care about it?

Already covered this in answers to previous questions.

33. Describe the modeling hypothesis and catharsis hypothesis concerning the effects of violent television on behavior.

Modeling hypothesis: people learn by watching others. Thus, the modeling hypothesis predicts that violent TV would lead to higher levels of aggression. Comes from social learning theory (Bandura and Walters).

Catharsis hypothesis: people drain off their stored, pent-up aggressive energies. Thus, the catharsis hypothesis predicts that violent TV would lead to lower levels of aggression. Comes from Aristotle (the psychological effects of drama) and has its more modern variation in Freudian theory.

34. What is the conclusion of the correlational studies of Eron (1963) regarding the effects of television on behavior?

There was a positive correlation between amount of TV watched and peer ratings of aggression among 875 Grade 3 students. This is consistent with the modeling hypothesis, not with the catharsis hypothesis.

35. What is the conclusion of the experimental studies of Bandura et al. (1961)?

Those who watched the aggressive model were significantly more aggressive toward the bobo doll than those who watched the non-aggressive model. Thus this study supports the modeling hypothesis.

36. How would you critique the Bandura et al. bobo doll study on the basis of external validity?

If you are trying to generalize the Bandura et al. study to real TV and real aggression among people, then:

(1) Independent variable: a videotape of an adult beating up on a bobo doll or playing with tinkertoys is not similar to a real TV program; (2) dependent variable: beating up on a bobo doll might not be the same as beating up on a real person.

37. How would you critique the Bandura et al. bobo doll study on the basis of internal validity?

If the claim is made that it was exposure to the aggressive content alone that caused the differences in aggression in the two conditions, then there is a possible confound (which would reduce the internal validity of the study): activity level was also (inadvertently) different in the two conditions. Specifically, in the bobo doll videotape, the model was also active, jumping around, etc., compared to the tinkertoy videotape. Thus, a possible alternative explanation was that activity level may have been at least partly responsible for the differences in the two conditions: maybe jazzing kids up leads them to be more aggressive. (I'm not sure I really believe this, but at least it is somewhat plausible.)

38. How did the Liebert and Baron study do better than the Bandura et al. study in external validity?

(1) Independent variable: showed a real TV program (The Untouchables in the aggressive condition); (2) dependent variable: they had the kids believe that they were either hurting or helping a real child in another room. Both of these differences made the Liebert and Baron more generalizable to the real world in addressing the question of whether TV violence could lead to real aggression among kids.

39. How did the Liebert and Baron study do better than the Bandura et al. study in internal validity?

They controlled for activity level by having the nonaggressive (control) program = an exciting sports sequence. In so doing, activity level could no longer explain the difference in aggression between the two conditions because it was (roughly) the same in the two conditions.

40. * Is there a causal relationship between violent television and aggressive behavior, according to reviews of the existing literature? Is it strong?

Yes--reviews of the literature support the conclusion that there is probably a causal relationship, although a weak one. On the other hand, even a weak effect could represent a societal problem when it is multiplied by 300 million North Americans.

41. What is the blood-brain barrier? And where is it?

The BBB protects the CNS from the rest of the body (and the rest of the body from the CNS). It is basically everywhere that the CNS interacts with the rest of the body--at the capillaries. Two physical aspects of the BBB that make it difficult for substances to get through: (1) tighter spaces between epithelial cells that line the space between the capillaries and the CNS, (2) different (more restrictive) active transport mechanisms that shuttle substances between the capillaries and the CNS. So, the BBB is not in a single location.

42. What does it mean that the blood-brain barrier is "selectively permeable"?

Some substances can pass through and others cannot.

43. Name one place where the blood-brain barrier is more permeable. Why is this a good thing?

Area postrema, which controls vomiting. That is a good thing because you want the area that controls vomiting to be more sensitive to the presence of possibly harmful substances in the bloodstream. Greater possibility of ejecting those substances.

44. What is myelin? How does it help the neuron?

Myelin is a protein that surrounds the axons of (some) neurons. It helps insulate the neuron, thus allowing the nerve impulses to travel down the axon with greater efficiency. Also helps insulate the neuron from the potentially interfering signals from other neurons.

45. Describe one theory about the causes of multiple sclerosis that involves the blood-brain barrier.

MS involves demyelination of neurons that are involved in motor control. One theory is that something (possibly a slow virus) has weakened the BBB and that the body's lymphocytes (white blood cells) have entered the CNS and attacked the myelin of those neurons. However, new evidence suggests that MS is not just a demyelinating disease--that in MS patients, there is actually neuronal death. So it is still unclear whether the demyelination is central to MS, or a consequence of some other disease process that causes neurons to die.

46. What is the difference between the central nervous system and the peripheral nervous system?

See your book for an excellent description.

47. Describe the distinction between the sympathetic vs. parasympathetic nervous systems.

See your book for an excellent description.

- 48. What does it mean that the sympathetic vs. parasympathetic nervous systems are “antagonistic”?**
They work in opposite directions. For example, the sympathetic NS accelerates heart rate and the parasympathetic slows it down. It is through the simultaneous push and pull of these two nervous systems that the body achieves homeostasis--balance. That’s how body temperature, heart rate, respiration, and virtually every bodily function operates within a (sometimes narrow) range.
- 49. What are the parts of a neuron? And describe the function of each.**
This is covered very well in Myers.
- 50. What are sensory neurons? Motor neurons? Interneurons? (which is afferent and which is efferent?)**
This is covered very well in Myers. Sensory = afferent; motor = efferent.
- 51. How do neurons transmit their messages from one to the other?**
Neurotransmitters. The neurons don’t actually touch but rather the pre-synaptic neuron sends neurotransmitter into the synapse where it is received by the receptors of the post-synaptic neuron. This process is well covered in Myers.
- 52. What is the nerve impulse (action potential)? What triggers one? And what specifically happens? (remember the toilet flushing analogy)**
This is covered very well in Myers, but here is my additional comment: when excitatory input from the upstream neurons is greater than the inhibitory input beyond a threshold, the nerve impulse is triggered. The toilet analogy: when the “excitatory input” from your hand’s pressure on the toilet handle is greater than the threshold for triggering the flush, the toilet does indeed flush. And it flushes the same whether you are gentle or forceful on the handle. Then there is a refractory period--that is, immediately after the toilet flushes, no amount of banging on the handle will cause the toilet to flush again (same with the neuron--there is a refractory period--about 0.3 msec--in which the nerve has to recover from its previous impulse).
- 53. What part of the neuron “summarizes” the excitatory and inhibitory input from other neurons?**
Axon hillock
- 54. If the nerve impulse is always the same magnitude, how do you experience intensity of sensation? (there are two main methods)**
1. More frequent firing; 2. Greater number of neurons firing.
- 55. What is the role of neurotransmitters in synaptic transmission? (Know the neurotransmitters I mentioned in lecture--acetylcholine, dopamine, glutamate, GABA, and serotonin)**
See the neurotransmitter handout.
- 56. What does the suffix “-ergic” as in “dopaminergic” mean?**
That the neurotransmitter is associated with this neuron. Thus, dopaminergic neurons are those that are associated with dopamine as a neurotransmitter.
- 57. Describe the events that take place during synaptic transmission.**
Covered very well in Myers.
- 58. What kinds of neurons in the cerebral cortex are killed off in Alzheimer’s Disease?**
Cholinergic neurons (those with acetylcholine as the neurotransmitter)
- 59. What is the relationship between aluminum and Alzheimer’s Disease? (and how is this related to the correlation-causation distinction?)**
Some evidence that aluminum collects in the brains of AD patients. Why? Could be that aluminum causes AD. But because this is correlational, it could be that AD causes aluminum to collect in the brain or that there is some spurious relationship between AD and aluminum in the brain. Some suggestion that it might be AD causes aluminum to collect in the brain and thus we shouldn’t avoid aluminum.
- 60. What kinds of neurons are killed off in Huntington’s Disease?**
GABA-ergic neurons in certain motor areas of the brain.
- 61. What kinds of neurons are killed off in Parkinson’s Disease (and in what part of the brain)?**
Dopaminergic neurons in the substantia nigra
- 62. Parkinson’s disease is characterized by a particular kind of motor problem—what is it?**
Particular problems with initiating and stopping movement.
- 63. Why can’t we just give Parkinson’s patients dopamine pills?**
Dopamine doesn’t pass through the BBB.

64. What is L-DOPA? How does it work? And how does this confirm the role of dopamine in Parkinson's Disease?

L-DOPA is the immediate precursor for dopamine (just one chemical synthetic step before dopamine). So if you give L-DOPA, it passes through the BBB where in the brain that chemical synthetic step takes place to yield dopamine. Giving L-DOPA helps PD patients; thus, this confirms the importance of dopamine in PD.

65. Why isn't L-DOPA a cure for Parkinson's Disease? (Why does it lose its effectiveness over time?)

L-DOPA makes more dopamine available for use by those dopaminergic neurons in the substantia nigra. But whatever is causing the death of those neurons continues and thus, over time, there are too few dopaminergic neurons around to use that increased dopamine that is present from L-DOPA treatments.

66. * How is dopamine implicated in schizophrenia?

67. * What (tentative) evidence do we have that schizophrenia and Parkinson's Disease may both be related to problems with dopamine?

68. What does it mean when I say that drug delivery is the basic problem of treatments for neurotransmitter-based diseases?

My car analogy: if your car needed oil, you wouldn't pour buckets of oil over the top of your car because none of it would get to the precise location where that oil is needed. Believing that the deficiency of a neurotransmitter system could be helped by simply giving the patient more of that neurotransmitter is like dumping your car with buckets of oil.

69. How does Prozac work as an anti-depressant? (that is, what neurotransmitter system is affected, and how?)

It is known that serotonin levels are associated with mood. So for those who are depressed, serotonin levels are lower than they should be. Thus, one theoretical method for helping depressed people would be to make more serotonin available. There are many methods that potentially could work (serotonin agonists, making the serotonin receptors more receptive) in theory, but some are practically impossible and some are deadly. Prozac is a selective serotonin reuptake inhibitor. When the presynaptic neuron releases neurotransmitter into the synapse, it is not a passive process: that neuron immediately starts sucking the neurotransmitter back into itself, a process known as reuptake. SSRIs like Prozac work by slightly inhibiting the reuptake process, thus allowing the postsynaptic receptors slightly more time to absorb more of the serotonin in the synapse. This happens very very quickly (on the order of 1/10,000th of a second). But SSRIs have fewer side effects because they are selective; that is, they only affect serotonergic neurons. In comparison, other anti-depressants involve other neurotransmitter systems besides serotonin, which leads to complications, some of which are serious.

70. How do SSRIs (selective serotonin reuptake inhibitors) help make more serotonin available at the synapses of serotonergic neurons?

See #69.

71. Review the parts of the brain. (Three basic divisions of the brain...)

Brainstem, limbic system, cerebral cortex. See Myers.

72. What does the reticular activating system (also known as the reticular formation) do?

The RAS (also known as the reticular formation) is a structure that connects parts of the brainstem to the thalamus. Associated with arousal. See Myers.

73. What is the cerebellum responsible for?

Coordinated movement. See Myers.

74. What does the thalamus do?

The switchboard of the brain: lots of projections into the thalamus, and lots of projections from the thalamus to the cerebral cortex. See Myers.

75. What does the amygdala do? What happens to the recognition of food aversions when the amygdala is damaged (the Garcia effect)?

Amygdala is associated with aggression and memory for significant stimuli, such as memory for food aversions. Garcia effect: (1) give a rat a food that it has never tasted; (2) make that rat sick 24 hours later from radiation (so that the actual source of the sickness is not the food); (3) rat will never touch the food again. This demonstrates that the rat has learned a single association 24 hours after the original stimulus (the novel food) was presented. This is a pretty exceptional phenomenon in rats. Now--if you remove the amygdala, this Garcia effect goes away, which demonstrates that memory for food aversion is controlled by the amygdala.

76. What is the Garcia effect?

See #75.

77. What is the hippocampus? And what is its primary function?

Memory (particularly short-term to long-term transfer of declarative memory). See Myers.

78. What happens when you have severe hippocampal damage? (Videotape on Clive Wearing, the musician whose hippocampus was destroyed by encephalitis.)

This is well-covered by the two videotapes on Clive Wearing and in the Myers book.

79. What is the difference between declarative memory and procedural memory? Which memory was damaged in Clive Wearing (and others who suffer from bilateral hippocampal damage)?

Declarative memory: memory for what; procedural memory: memory for how. Declarative memory was destroyed in Clive Wearing, who suffered from severe anterograde amnesia. See the handout on Clive Wearing, which accompanied the videotapes.

80. What is the hypothalamus? What activities does it control?

Pretty small structure of the brain associated with motivations and drives. The 4 Fs: feeding, fleeing, fighting, and fsex.

81. What is the nucleus accumbens? What does it control? What neurotransmitter is associated with it?

Small structure that has connections with the hypothalamus--the pleasure center of the brain. Specifically, it is the dopaminergic pathways of the nucleus accumbens that are important.

82. Describe the paradigm that James Olds invented to determine (1) where in the brain is the site for reward, and (2) which substances have the potential for addiction.

Olds did studies that implanted electrodes in various parts of a rat's brain. Then allowed the rat to self-stimulate (that is, to send an electrical signal to that electrode by pressing a bar). Eventually found a place in the brain where the rat would self-stimulate for very long periods of time. Obviously found a pleasure center in the brain. Then made it difficult for the rat to get to the self-stimulating bar (e.g., putting a electrified grid between the rat and the bar). Rat would traverse that grid over and over again. Indicates that the rat really wants to stimulate that area of the brain (the nucleus accumbens).

Could do studies involving substances to measure how much a rat (or other animals) would want that substance by making it hard to get to that substance. Nice measure of that substance's addictive potential.

83. Compare the rat's desire for seeking self-stimulation (through drugs) to the desire for food.

The rat won't eat or drink or do anything else except press that self-stimulation bar.

84. What is the neurotransmitter that may be responsible for the self-stimulation effect.

Dopamine (but specifically the dopaminergic pathways of the nucleus accumbens).

85. What are endorphins?

Neurotransmitters that are essentially identical to opiates (opium, codeine, morphine, heroin). So you can refer to endorphins as internal opiates. They are associated with pain relief and pleasure.

86. What is the neurochemical explanation for why withdrawal is so painful for opiate addicts?

If an addict is taking external opiates (e.g., in the form of heroin), the brain makes less endorphins. So if an addict goes off heroin, there are lower levels of endorphins available--and life becomes very painful.

87. There is another neurochemical effect of alcohol addiction--what is it?

Chronic alcohol use leads to a suppression of calcium channel activity. Calcium channels are a mechanism by which the presynaptic neuron spits out neurotransmitter into the synapse. In response to chronic suppression of calcium channels due to alcohol use, over time, more calcium channels are created. But now if the alcohol addict stops taking alcohol, suppression is lifted, but there are now more calcium channels spitting out more neurotransmitter--too much. This leads to a variety of very unpleasant physical symptoms (e.g., delirium tremens--DTs).

88. How does the size of the cerebral cortex relate to position on the "evolutionary ladder"?

The higher you go on the evolutionary ladder, the greater the proportion of the brain that is cerebral cortex

89. Know the four lobes of the cortex and what their function is (mostly in Myers).

Four lobes: frontal, parietal, occipital, temporal. See Myers.

90. What are the frontal lobes responsible for?

Higher mental functioning, reasoning, some aspects of personality (remember Phineas Gage from the Myers book and Clive Wearing).

91. What is the corpus callosum?

Very dense bridge of neurons connecting the left and right hemispheres.

92. What was Descartes' conception of physiology?

He had a mechanistic conception of the body: the body is a machine. (Deus ex machina).

93. In Descartes' view, how did memory and learning occur? How about sensory input?

Descartes believed that neurological/neuropsychological events occurred from the passage of cerebrospinal fluid through tiny little tubes and valves. So memory and learning would occur when the tubes holding that particular memory or learning situation would have valves leading into those tubes that were slightly open, allowing fluid to flow more easily into those areas. Sensory input occurred when fluid would flow away from the sensory location (e.g., finger) into the spinal cord. And then if the sensory input was dangerous (e.g., the proverbial finger over a candle flame), the sensory input would be faster, and the tubes and valves going from the spinal cord to the finger's muscles would go faster, resulting in a quick withdrawal of a burnt finger.

94. What was the reflex, according to Descartes?

See #93.

95. What was Aristotle's view about the importance of the brain?

Aristotle believed that the brain was a minor organ, mostly responsible for condensing the vapors of the body's humours.

96. Describe Gall's theory of the brain and his theory of phrenology.

Gall believed that there were very specific mental faculties that were represented in specific locations in the brain. For example, he believed that there was a place in the brain where the faculty of "hope" was located, or "parental love" or "self-esteem." Second--he believed that if an individual was well-endowed with a certain faculty, that area of the brain would be bigger. Third--he believed that if your brain was bigger in a certain area, it would push out the skull so that you could feel it. Thus, you could tell an individual's mental faculties by feeling for bumps on the head.

97. What were Gall's mistakes in the theory of phrenology?

There is no such specific localization in the brain corresponding to Gall's theory. Your brain wouldn't grow bigger if you developed more of a certain faculty. And certainly, your mushy, soft brain couldn't push out your cranium, perhaps the hardest bone in your body.

98. Describe Flourens' experimental methods in studying the functions of the brain.

Flourens was a skilled surgeon who used experimental methods. Specifically, he conducted ablation studies in which he would selectively destroy parts of the brain of an animal (the independent variable was the specific area of the brain destroyed) to see what changes would be observed in the behavior of the animal (the dependent variable).

99. Describe how Flourens tested Gall's ideas through ablation techniques. What did he find?

Flourens decided to test Gall's idea that an area of the brain at the lower back of the head was the area of "amativeness", or sexual responsivity. So he cut the brain at that location in a dog. What he got instead of changes in sexual responsivity was the classic signs of a cerebellar lesion--loss of coordinated voluntary movement. Gall was wrong about the function of that area of the brain.

100. What is action propre? What is action commune?

Action propre--specific action (localization in the brain)

Action commune--the brain acting as a whole

101. What was Flourens' conclusion about localization in the brain?

Although there was some action propre (some degree of localization), it was mostly action commune.

102. What did Broca's research conclude about the localization of speech?

See #103

103. Who was "Tan" and why is he (his brain) important in the history of neuropsychology?

Tan was a patient of Broca's who had lost his speech 21 years earlier. Tan could understand language but could not produce language. When Tan died, autopsy revealed a oval area of damage to the left frontal lobe, just anterior to the motor strip associated with mouth/tongue movements. This is known as Broca's area. It makes conceptual sense that the damage would be there--it's not in the motor strip itself because then Tan wouldn't be able to move his mouth/tongue. It's in an association area next to that part of the motor strip--where the production of language is located. Patients who have damage to Broca's area can understand language but they can't produce language. This is known as motor aphasia (or Broca's aphasia).

104. What is aphasia?

Neurological disorder in which language is impaired in some ways. Several different types.

105. Where is the motor strip? Where is the sensory strip? How did Fritsch and Hitzig discover them?

Motor strip is in the frontal lobe, just anterior to the central sulcus (fissure), which separates the frontal lobe from the parietal lobe.

106. What are association areas? How do they relate to the sensory areas?

Association areas are areas of the cerebral cortex that are responsible for more higher-order brain functioning. For example, Broca's area is not the mouth and tongue regions of the motor area itself, but rather next to it, where some of the higher-order aspects of moving the mouth and tongue are represented--namely the production of language. Same with the relationship between association areas and the corresponding sensory areas.

107. What did Wernicke discover about the comprehension of speech? Where is it?

Wernicke had patients who had problems with the comprehension of language. Their speech was fluent (no problems producing words), and their syntax was intact (that is, they spoke in grammatical sentences), but the semantics (meaning) was all off. Brain damage was in the temporal lobes, in the association areas for language memory.

108. Distinguish between motor aphasia (Broca) and sensory aphasia (Wernicke).

See above. In addition, sensory aphasics show more deficits--they can't use language properly in any way. Moreover, they show a real curious lack of awareness that they have a problem at all.

109. What was Lashley's goal? What is the engram?

Lashley wanted to find the engram, the hypothesized area of the brain associated with learning and memory.

110. What did Lashley discover about the localization of learning?

Mostly the brain acting as a whole (see Flourens). Certainly didn't find the engram.

111. What is equipotentiality? What is the Law of Mass Action?

Equipotentiality means that all areas of the brain have the same potential for a given function--all parts of the cerebral cortex can carry out the functions lost if another part of the cortex has been destroyed. This is NOT true for specific functions of the brain such as language, movement, but seemed to be true for learning and memory in Lashley's research. The Law of Mass Action means that the more brain you destroy, the worse the performance (on a learning task), and it doesn't much matter where the destruction occurs. Note that this is an overstatement, and it doesn't apply for all (or even most) of the functions of the brain (e.g., language is very specific and localized). Lashley used these terms in his research on the search for the engram.

112. What did Penfield discover in his work on epileptics?

Worked with severe epileptics. Conducted surgery to alleviate their symptoms by identifying the area of the brain associated with auras (those sensations experienced by some epileptics just before an attack). Opened up the skull and stimulated areas of the brain to recreate the aura. Would remove the area of the brain associated with that aura. Led to significant improvement in many epileptics. While he was in the brain anyway, Penfield stimulated other areas of the brain--led to interesting results. Don't worry about this material--some of it is described in Myers.

113. Describe the Rosenzweig experiments on how a rich environment affects the brain. What is the significance of the increase of acetylcholine in the brain of the rat who has been in a rich environment?

Rosenzweig conducted experiments with baby rats. He randomly assigned newborn rats to one of two conditions:
(1) impoverished environment: plain cages in dimly illuminated, quiet room; nothing to do;
(2) rich environment: running wheels, ladders, slides, and toys. Changed toys everyday to change the environment. More learning could take place.

He then "sacrificed" the rats and looked at their brains. Many differences:

Rich environment rats: thicker cortex, better capillary supply, more glial cells, more ACh in the cortex; more dendritic branching, etc.

The greater levels of ACh in the cortex is significant because ACh is implicated in memory: remember that Alzheimer's disease is associated with the death of cholinergic (ACh) neurons in the cerebral cortex)

114. Describe Greenough's findings on putting middle-aged and elderly rats in enriched environments.

Greenough did the same kinds of studies as Rosenzweig but with middle-aged and elderly rats. Found similar results (so rich environments can stimulate and enhance the brains of older rats as well!).

For example, he found that there were 2,000 additional synapses per neuron in the rich environment rats. That translates to trillions of new connections.

115. What does it mean to say that the brain has remarkable plasticity?

The brain is very responsive to the environment. Stimulation leads to positive effects. But if the brain is exposed to negative environment, this can lead to negative effects on the brain.

The remaining questions will NOT be covered on Midterm 1 (they will be covered on Midterm 2).