NTNU
Det skapende universitet

Psykologisk institutt

Eksamensoppgave i PSYPRO4412 - Anvendt og klinisk kognitiv psykologi

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Tlf.: 73 59 19 60

Eksamensdato: 04.12.2015

Eksamenstid (fra-til): 09.00-13.00

Hjelpemiddelkode/Tillatte hjelpemidler: Ingen

Annen informasjon:

Målform/språk: Bokmål

Antall sider: 2

Antall sider vedlegg: 0

Kontrollert av:

Langsvarsoppgaver

(Velg 2 av 3, hver teller som 40% av den endelige karakteren)

Spørsmål 1:

Hva er "nevrofeedback"?

Kan man ved hjelp av dette gjøre ting som ville vært vanskelig eller umulig å oppnå kun ved å snakke med folk?

Spørsmål 2:

Hvordan ville du designe et modifiseringsprogram for oppførsel?

Hvilke teoretiske avveiinger vil legge føringer for ditt design?

Spørsmål 3:

Hva er rekonsolidering og hvorfor har det en betydning?

Kortsvarsoppgaver

(Velg en av to, svaret teller 20% av den endelige karakteren)

Spørsmål 4:

Hva er attribusjon av personlig handling(agency), og hvorfor er det relevant å ha vrangforestillinger om kontroll?

Spørsmål 5:

Forklar «the drift-diffusion model» for det å velge mellom to alternativer.
Hva slags parametere kan hentes ut fra reaksjonstid distribusjoner?

Hva er den kliniske relevansen av slike parameteranslag?

Long questions (Choose two of three, each counts as 40% of the final grade)

Question 1: Hva er nevrofeedback? Kan de gjøre hva er vanskelig eller umulig å gjøre ved å snakke med folk? What is neurofeedback? Can it do anything that would be difficult or impossible to achieve by talking to people?

Eksamensoppgaven tar utgangspunkt i en labøvelse studentene har gjennomgått. 10 har gjennomført en qEEG-måling før og etter nevrofeedback treninger, der resten av kullet har deltatt som observatører. I tillegg har de to artikler om nevrofeedback som pensum.

Drøftinger har vært rundt hvilke mekanismer som nevrofeedback berører, altså operant betinging av hjernebølger som i dette tilfellet har ført til forbedret evne til konsentrasjon. I tillegg har studentene deltatt i diskusjoner på i hvilken grad nevrofeedback kan føre til bedringer om det er snakk om lidelser som ikke har et tydelig fysiologisk grunnlag, som ved traumatisering.

Det er ønskelig at besvarelsen tar opp muligheten til å endre uttrykket av hjernebølger, at det er betydningsfullt at hjernebølger kan framstå med forskjellige frekvenser og at det er vist empirisk at endring av hjernebølgeuttrykk gir funksjonelle bedringer både i normale personer og i pasienter. I tillegg bør det komme en drøfting av treningseffekter både der det er et fysiologisk grunnlag for en lidelser og der det ikke er det. Akkurat her er det få absolutte svar, så de er oppfordret til å drøfte og eventuelt å spekulere.

Question 2: How would you design a behavior modification programme? What theoretical considerations guide your design?

The basic problem with inducing behavior change is relapse, and one important cause of relapse is that behavior modification training often does not affect the original learning that causes undesirable behavior, but causes new learning. That means there are competing and conflicting pieces of information in memory. The new learning is often context-specific, and relapse may occur when returning to the context of original learning. That would be an example of ABA renewal, where A is the original context and B is the context in which the learning occurs that is supposed to change behavior. Students may choose to define and explain AAB, ABA and ABC renewal as well as spontaneous recovery, reinstatement, rapid reacquisition and resurgence, or they may choose just to mention these problems and focus on how to address them. There are several approaches.

One possibility is to accept that new learning is context-specific, but try to make it as effective as possible, and to get around context specificity to some degree by training in multiple contexts. Further, if extinction proceeds very gradually, for example in extinguishing fear conditioning by starting with the least scary situations, there is the risk that reduced fear is based mostly on the non-associative process of habituation. If instead mildly and quite scary situations are mixed, then prediction error is kept high enough to increase learning rate and speed up new learning. It also helps if subjects are encouraged to make very specific predictions so that it is clear when those predictions fail. Prediction errors may also be increased by first extinguishing responses to several conditioned stimuli separately, then combining those stimuli, by removing safety signals (when treating phobias, cognitive treatments that reduce anxiety would also reduce prediction error and should therefore only be given *after* extinction training), and by occasional CS-US pairings. Generalisation of the new learning is encouraged by varying the conditioned stimuli and the contexts in which they are presented.

An alternative view is that a sudden change in the relationship between conditioned and unconditioned stimulus acts like a context change and that this is what triggers new learning. If instead the probabilistic relationship is changed more gradually (though presumably not so gradually

to risk relying on habituation alone), then the original association is modified. This avoids the context specificity of new inhibitory learning and so avoids renewal and reinstatement. This approach seems to contradict the idea of maximizing prediction error, yet the emphasis is on a gradual change, not on the magnitude of prediction error.

Finally, reconsolidation may be employed. Within a time window more than 10 minutes but less than 6 hours after retrieval, a memory may be labile and thus modifiable. The memory may then be modified through extinction training within that time window, or a range pharmacological interventions, most commonly administration of cortisol. As in the case of gradual change of the CS-US relationship, targeting the original memory avoids the effects of context specificity such as renewal, spontaneous recovery and resurgence.

Question 3: How reliable is memory, and what mechanisms do you know that could produce false memories?

Geraerts et al. divided memories of childhood sexual abuse into continuously remembered (there was never a time when the person reporting abuse had forgotten the abuse) and discontinuous, and they divided the discontinuous memories into those remembered within or outside of therapy. Geraerts et al. do not mention whether the therapy in question was specifically recovered memory therapy or a broader range of therapies. In this study, *none* of the memories recovered in therapy was corroborated by independent evidence, while memories recovered out of therapy were as likely to corroborated as continuous memories (those that had never been forgotten). The best predictor of the existence of corroborating evidence was being surprised by the memory. Memories that were corroborated also tended to be recovered suddenly, rather than gradually.

Shaw and Porter interviewed subjects three times, asking them both about an emotionally salient event that (according to the subjects' parents) they really had experienced, and about a fictitious crime. Shaw and Porter were able to persuade 70% of subjects that they had committed a crime serious enough that police had gotten involved. Their interviews used tactics that had elicited false confessions in previous research, such as presenting false evidence, social pressure, building rapport, suggestive retrieval techniques including guided imagery, leaving pauses for the subjects to fill, and implying that experimenters knew further details.

The finding that corroborated discontinuous memories were recovered more suddenly and were more surprising than uncorroborated (and therefore more likely false) memories suggests that false memories are built up slowly. There are three candidate mechanisms within the pensum. One is reconsolidation. Hupbach et al demonstrated that episodic memory can be altered through reconsolidation. They showed people a list of objects on a Monday. On Wednesday, the reconsolidation group was asked to remember what they had done on Monday. Then they were shown a second set of objects. One control group saw the same second set of objects, but without being reminded of the previous set. Those two groups returned on Friday, joining the second control group. The experimental group encoded more Wednesday objects along with the Monday objects of which they had been reminded than the control group without the reminder. The Monday & Friday only group provided a baseline of spontaneous intrusions without exposure to the Wednesday objects. The intrusions were asymmetric. The experimental group did not include Monday items in their recall of the Wednesday items. The increased intrusions were of Wednesday items seen at a time when the Monday items were re-encoded after the reminder. Reconsolidation this provides a mechanism by which memories may altered, rather than merely new memories being established that may compete with the old ones. That matters in the context of false memories. Depending on the circumstances in which a false memory is established, the original information may be overwritten.

An alternative method of altering an existing memory is to change the probabilistic relationship

between a conditioned and an unconditioned stimulus gradually. The theory behind this approach is that a sudden change in the relationship between conditioned and unconditioned stimulus acts like a context change and triggers new learning, represented in a separate, parallel association. Gradual change in the relationship targets the original association. It is unclear whether this would generalise from conditioning to episodic or semantic memory.

A sudden change in the CS-US relationship is more likely to generate new, parallel associations. In the case of conditioning, there would be no explicit information regarding the source of the information, and it would be impossible to tell directly which is the original memory.

The pensum in this course does not contain any studies that attempt to establish how much each of these mechanisms may contribute to any particular false memory. All that matters here is that students understand the distinct mechanisms, that two of them modify and original memory, while one establishes a new memory that is stored in parallel with the old, and that paper in the pensum only provide evidence for reconsolidation affecting episodic memory.

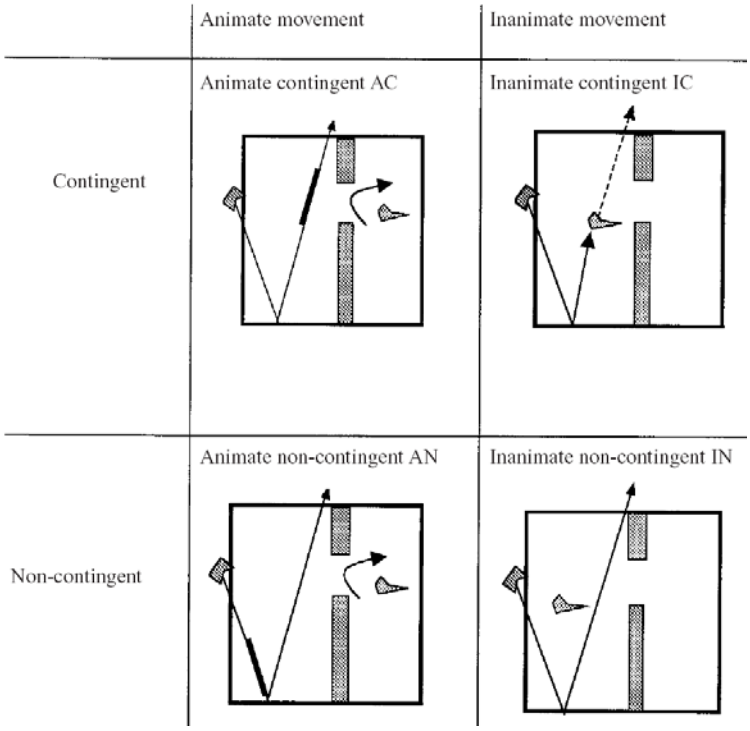
Short questions (Choose one of two, each answer counts as 20% of the final grade)

Question 1: Hva er ″attribution of agency″, og hvorfor er det relevant for kontrollvrangforestillinger? (What is attribution of agency, and why is it relevant to delusions of control?)

Attribution of agency is explaining events as the consequences of the actions of an agent, that is someone who has intentions. Frith argues that excessive attribution of agency is needed to explain delusions of control (the belief that someone other than the patient controls the patients actions or thoughts) because problems with the forward model are not a sufficient explanation.

A forward model predicts the motoric and sensory outcomes of motor commands. A large enough deviation from that prediction can reasonably be interpreted as indicating an external causal factor that affects the outcome. If the forward model is faulty and produces larger prediction errors than justified, then deviations from prediction that actually are small and random can be interpreted as indicating an external influence. The anarchic hand phenomenon, caused by lesions to supplementary motor cortex or anterior corpus callosum, demonstrates that problems with the forward model can only explain the feeling of not controlling one's own actions, but not the feeling that someone else controls one's actions. Anarchic hand patients feel one of their hands has a will of its own, but not that someone else controls it.

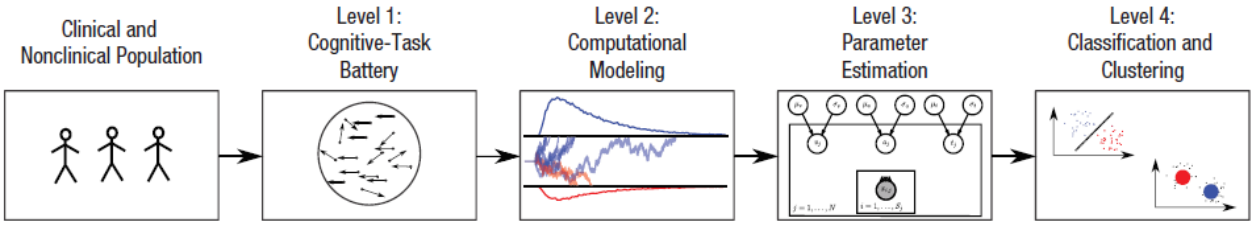
Excessive attribution of agency means seeing evidence of intentional action where the general population sees less or none. The most intuitive data presented to the students comes from an experiment on moving geometric shapes. Subjects were shown all four combination of animate and inanimate shapes (they move under their own power or only passively, like billiard balls) and contingent (the shapes interact) versus non-contingent.



The question was whether there would be an interaction between animation, contingency and diagnosis. The difference between inanimate contingent and inanimate non-contingent provides a baseline, and was similar in delusional patients and controls. However, delusional patients perceived no difference between animate contingent and animate non-contingent conditions, attributing intentions to the animate-non-contingent movements that controls did not. When this exaggerated attribution of intention to some outside agency is combined with not feeling in control of one's own movements or thoughts, the result is to attribute control to someone else. That is a delusion of control.

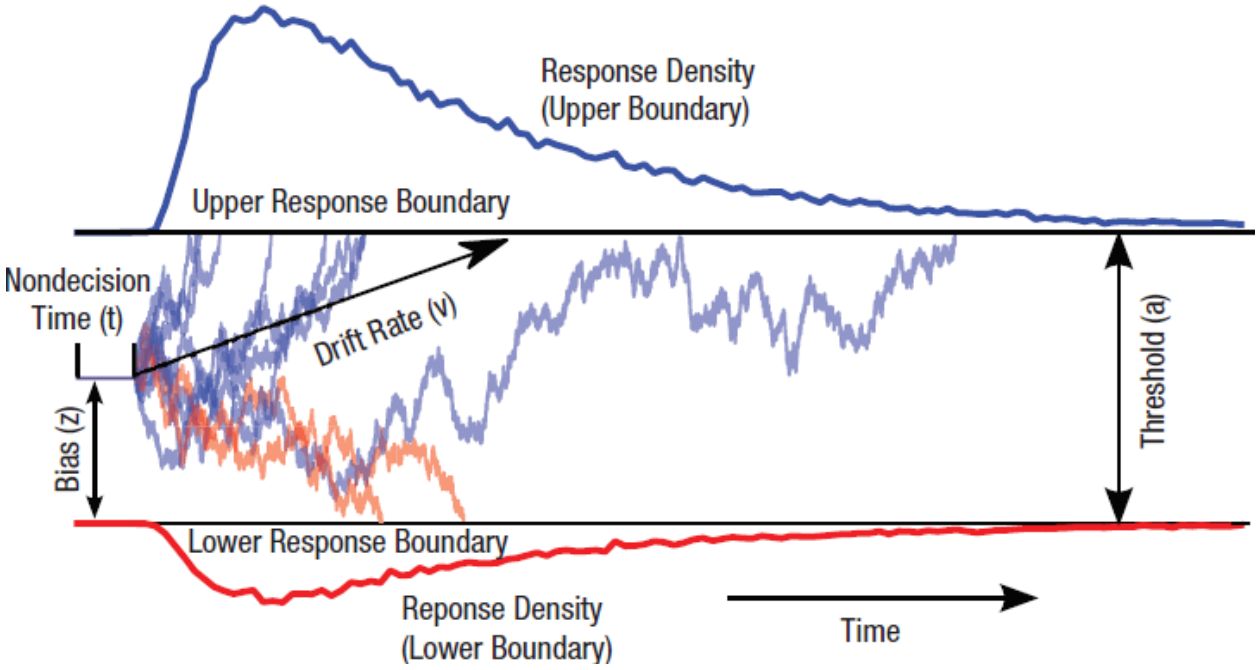
Question 2: Explain the drift-diffusion model of choosing between two alternatives. What parameters can be extracted from reaction time distributions? What is the clinical relevance of such parameter estimates?

The drift-diffusion model is a specific example of computational modelling applied to mostly perceptual tasks with two response alternatives, such as the dot motion in the second frame of this figure (Level 1):



The drift-diffusion model assumes that the decision variable starts between two decision boundaries, that noisy evidence for each alternative is added up over time, moving the decision variable to one or the other of the boundaries, and that a decision is made the moment the decision variable reaches one of the boundaries. From the distribution of response times, recorded separately for correct and incorrect answers, it is possible to reconstruct:

1) Nondecision time, which is how long it takes for information to reach the relevant part of the brain, and for the motor command to result in a button press.
2) The starting point for the decision process, called bias because it may be closer to one of the decision boundaries.
3) The drift rate, how quickly the decision variable changes.
4) The threshold, or distance between the decision boundaries.



Although Wiecki et al do not discuss this, it should be easy to work out that bias should respond to base rates and to costs and benefits. In the present example of the dot motion task, if movement up is more common than movement down, then average response time and proportion correct answers can be improved by moving the starting point closer to the upper response boundary. A faster drift rate would reduce response times without affecting proportion correct. In contrast, moving the decision boundaries closer together (changing the threshold) would speed up responses at the cost of reduced accuracy because noise in the system will have a relatively greater influence.

That response time can be affected by changes in several of the parameters of the underlying computation illustrates the task-impurity problem of a simple response time measurement. Estimating the computational parameters reduces such problems, and therefore should more cleanly separate clusters of people who are lumped together by the simpler measurement.