

The development of delayed response: Parallel distributed processing lacks neural plausibility

Stanislas Dehaene

INSERM U. 334, France

Abstract

Munakata's model of the A-not-B task provides an excellent fit to behavioral data from human infants. From a neuropsychological standpoint, however, its architecture is not very plausible. Dehaene and Changeux's (1989) neuronal model of delayed response tasks, while admittedly very simple, relies on identified features of brain architecture such as multiple hierarchical pathways, bistable clusters with sustained activity, and diffuse reward systems. Ways in which the insights provided by both models might be combined are discussed.

Eight years ago, Jean-Pierre Changeux and I isolated delayed response tasks as a rich domain where data coming from infant psychology, animal behavior, lesion and neurophysiological studies were available, and which therefore seemed well-suited for cognitive neuroscience modeling. The neuronal model that we developed (Dehaene & Changeux, 1989; hereafter D&C) used a simple, neurally inspired architecture to successfully account for the basic features of the data: the failure of young infants and primates in delayed response tasks, including the systematic A-not-B error; the successful performance of older infants and primates; the failure of subjects with lesioned prefrontal cortex; and the long-lasting, stimulus-specific response of prefrontal cells during task performance.

Munakata's PDP model of the A-not-B task goes significantly beyond our initial model in mimicking behavioral data from human infants. It provides an excellent quantitative fit to behavioral experiments that probe performance as a function of delay length, age, and various subtle variations on experimental conditions, such as using multiple locations, distinctive covers, or no hidden toys. In this commentary, I would like to argue, however, that this impressive behavioral fit is achieved within a hybrid network architecture that conforms neither to the classical parallel distributed processing (PDP) framework, nor to data from neuropsychology and neuroanatomy.

Contrary to what the paper's title suggests, Munakata's model departs considerably from traditional PDP models. Although it is a stimulus-driven, input-output

network, two of its crucial features are, first, the presence of prewired connections between layers (typical PDP models start with random connections), and second, the modeling of age by a pure maturational parameter, the growth of synaptic strengths in the hidden layer (typical PDP models account for age effects by learning through exposure to a structured environment). Although this is sometimes occulted in the paper, these two features, which are critical to the excellent match between the model's behavior and the empirical data, endow the network with a considerable amount of innate, biologically-determined structure. Classical PDP learning contributes only to explain within-experiment changes in behavior. In the model, long-term changes in the representational capacity of the network are attributed exclusively to maturation. Thus, the philosophy of the model, of biological inspiration, is in radical contrast to recent constructivist approaches to infant development (Elman *et al.*, 1996; Quartz & Sejnowski, in press) and is actually more compatible with the nativist views that it purports to criticize.

Still, the model has not fully shed its PDP origins. Critically, its architecture, in which grasping, visual expectancy, working memory and reaching behavior all go through the bottleneck of a single layer of hidden units, is implausible. Munakata herself notes that prefrontal cortex lesions selectively affect delayed-response tasks while leaving reaching and grasping unimpaired (Diamond & Goldman-Rakic, 1989). Her model, however, is incapable of simulating such neuropsychological dissociations, which strongly suggest that

Address for correspondence: INSERM U. 334, SHFJ, CEA, 4 Place du Général Leclerc, 91401 Orsay cedex, France. Phone +33 1 69 86 78 73; Fax: +33 1 69 86 78 16; e-mail dehaene@shfj.cea.fr

multiple parallel, hierarchically organized neuronal networks contribute to task performance in animals and infants (Goldman-Rakic, 1988).

The purpose of the Dehaene and Changeux (1989) model of delayed-response tasks, precisely, was to propose a minimal neuronal architecture compatible with neuroanatomical and neuropsychological evidence. To close this commentary, I would like to suggest ways in which the two models might be reconciled. First, the D&C model was organized in several parallel circuits: a lower-level sensori-motor loop supporting grasping, a higher level layer of units with long-lasting firing supporting working memory, and two even higher layers encoding rules (allowing the network to perform either spatial delayed response or delayed matching-to-sample tasks) and rewards (allowing the network to change its behavior following erroneous trials). Eight years later, this basic architecture, though admittedly very simplified, still seems to capture important neuropsychological dissociations (Dehaene & Changeux, 1996). It would be easy enough to supplement Munakata's simple input-output network with a similar architecture. For instance, the addition of direct one-to-one connections from location units to gaze units and to reach units would provide a better account both of infant's initial eye movement and grasping abilities and of their dissociability from more complex delayed-response performance following brain damage in animals.

Conversely, the second layer of the D&C model, comprising self-connected memory units, might be made to follow Munakata's assumption of an increasing synaptic strength with age. This would allow the D&C model to reproduce the smooth empirical curves for performance as a function of delay and age, which are captured so well by Munakata's model. A minor difference here is that the D&C model used bistable memory units with a discrete switching on and off, while Munakata uses units with a smoothly decreasing level of activation associated with progressive forgetting. Munakata provides no argument in support of her view that discrete bistable units are biologically 'less plausible' than graded units. In vivo recordings of prefrontal neuronal activity in behaving monkeys during delayed-response tasks often show sharp onsets and offsets as well as fairly constant levels of sustained firing during the delay period, with occasionally decreasing or even increasing firing rates (e.g. Kojima & Goldman-Rakic, 1982). Munakata's hypothesis that errors on long-delay trials are due to the slow decrease of firing rates back to zero is not supported by neurophysiological evidence, which indicates all-or-none firing rates on error trials as well as on correct trials (Niki & Watanabe, 1976). Note that bistable units can readily explain the smoothly

decreasing curves for performance as a function of delay length, because the probability of switching back to the off state due to ambient noise increases smoothly with elapsed time (Dehaene & Changeux, 1989).

Finally, the D&C model, like subsequent models based on similar architectures (Dehaene & Changeux, 1991; Dehaene & Changeux, 1993; Dehaene & Changeux, 1996), relies heavily on the hypothesis that learning is driven by internal or external reward signals that provide clues as to the correctness of the current behavior. Munakata criticizes this notion, stressing that 'infants receive no reinforcement for attending to events during their presentation'. The misunderstanding, here, is based on a narrow reading of the word 'reward'. Monkeys trained in delayed-response task receive a genuine food reward. As for infants, the retrieval of the toy itself, the emotional signals sent by the surrounding adults, or even the achievement of a correct prediction as to the toy's location, may serve as internal rewarding stimuli. During complex reasoning tasks in adults, the computation of internal reward signals is essential to learning (Damasio, 1994). As the neurophysiological basis of modulation of learning by diffuse catecholaminergic reward systems is getting better known (e.g. Montague, Dayan, & Sejnowski, 1996), I strongly believe that models of development based purely on 'associations between perceptions, representations, and responses', which lie at the heart of Munakata's model, will have to be supplemented by mechanisms of learning by selection (Changeux & Dehaene, 1989) driven by multiple internal and external reward systems.

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On theory and modelling

Jean M. Mandler

University of California San Diego, USA

Munakata asks: why do infants perseverate in the AB task? It doesn't seem like a good idea to try to answer this question without considering the larger issue of perseveration in general. Certainly a good first hypothesis would be that infants perseverate for the same reason that adults do. Munakata disapproves of explaining infants' perseverative behavior as an 'ancillary deficit,' but adult perseveration is exactly that. For example, we move our wristwatch from one arm to the other; we know where it is but continue to look for it in the wrong place. Motor habits do not require attention, and if we are thinking about something else, tend to run off perseveratively. This is indeed an 'ancillary deficit,' not lack of knowledge of the location of the watch. (Knowledge of the watch's location is independent of the current behavior, but it isn't clear why it must therefore be 'disembodied'; indeed, I am not sure what Munakata means by that term.) The same kind of perseverative behavior can be found in both infants and adults. For example, after several trials of watching a toy train go along a track through a tunnel, both infants and adults learn to anticipate its coming out of the other end, as shown by their eyes moving to the tunnel's exit before the train appears. If the train stops in full view, both infants' and adults' eyes tend to move to the exit even though the train is still in front of them (Chromiak & Weisberg, 1981; Bower *et al.*, 1971). When infants show such perseveration they have been said to think

objects can be in two places at once, but this argument loses its force when adults display the same behavior.

However, Munakata implies that if she can model perseverative behavior in the AB task without calling upon 'disembodied' knowledge of the location of hidden objects, then one can assume that perseveration takes place without such knowledge. This is equivalent to saying there is only one way to skin a cat: e.g., if one can build a computer to play chess then that is the only way that chess is played. To demonstrate that independent knowledge of hidden objects is not necessary, Munakata builds a looking and reaching model designed to produce the AB error (much like building a chess playing machine). The model works quite well. Unfortunately, this does not tell us what infants do know about object location, which was the original goal of the AB task. The model shows that you can mimic various AB error phenomena by manipulating the frequencies of looking and reaching, but that leaves us in an agnostic position as to whether or not infants know there is an object hidden under a cover. (Indeed, the AB task is a terrible test of object knowledge because it involves so many different processes: knowledge about object properties, perseveration, memory, and proactive interference are all involved. Piaget erred in his conclusions about infant representation because he based many of them on this task; unfortunately, we continue to err in this regard today).

Address for correspondence: Department of Cognitive Science, University of California San Diego, La Jolla, CA 92093-0515, USA.