LETTER

Reply to Bäuml and Hanslmayr: Adding or subtracting memories? The neural correlates of learned interference vs. memory inhibition

Tomlinson et al. (1) proposed an interference model of the no-think paradigm, but Bäuml and Hanslmayr (2) replied that this account is inconsistent with neurocognitive results. In the no-think paradigm, learning to suppress a target memory produces a recall deficit regardless of the cues used to test memory. Previously, inhibition provided the only explanation of this cue-independent forgetting. The model by Tomlinson et al. (1) proposed an alternative explanation by assuming interference from newly learned associations between partially retrieved memories and the no-think response (e.g., sitting quietly for 4 sec). Tomlinson et al. (1) tested this interference model with a "quickly press-enter condition." As predicted, the press-enter condition produced as much forgetting as no-think training.

In their reply, Bäuml and Hanslmayr pointed out that the interference model requires learning during no-think trials but fMRI studies find less hippocampal activation during no-think trials than during retrieval trials (3, 4). Tomlinson et al. (1) provided accuracy feedback for all conditions and all trials throughout training to equate the opportunity to learn. Nevertheless, a fit of the interference model to the data revealed 31 times greater learning for retrieval trials than no-think trials ($R = 31.0$ vs. $O = 1.0$). Thus, the interference model is in agreement with neurocognitive studies reporting larger neural responses during retrieval trials than during no-think trials (3–5).

One study found lower hippocampal activity during no-think trials compared with a baseline condition (4). However, comparisons to hippocampal baseline conditions can be ambiguous (6). Because the interference model requires only a small amount of learning during no-think trials, this result is explainable if the hippocampal baseline condition included even a modest degree of off-task activation.

Another way to measure learning is to use recall performance to "predict" neurocognitive activity during training. In this "subsequent memory" analysis, one can either select people who forgot more or select forgotten words. In a study that selected people, forgetters had progressively lower no-think event-related potentials (ERPs) over the course of no-think training compared with retrieval ERPs (5). Conversely, in fMRI studies that selected words, forgotten no-think words had greater hippocampal activation than remembered no-think words during no-think trials (3, 4). As expected by the interference model for the formation of new associations, this was particularly true at the beginning of no-think training. Finally, when the selection was over both people and words, the hippocampal increase was even greater for forgetters (3). These fMRI results support the interference model by revealing increased hippocampal no-think activity for subsequently forgotten words.

In proposing the interference model, Tomlinson et al. (1) did not deny the existence of inhibition as a neural mechanism. Consistent with the finding by Hanslmayr et al. (5) of reduced ERP responses in anticipation of a no-think trial, perhaps the role of inhibition (or perhaps inattention) is to reduce learning during no-think trials. Regardless of what role inhibition plays, the question of interest is whether no-think forgetting occurs through selective reduction of old memories or through learned avoidance. We hope that the interference model will motivate new behavioral and neurocognitive studies that distinguish between these alternatives.

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