Our aim was to test the hypothesis that common network switching mechanisms apply across tasks with varying cognitive demands and differing stimulus modalities. If confirmed, our findings would provide insights into fundamental control mechanisms in the human brain.

Results
We describe findings from Experiment 1 in the first three sections. Convergent findings from Experiments 2 and 3 are described subsequently.

Activation of CEN and SN, and Deactivation of DMN During Auditory
Event Segmentation. As reported previously (19), we found robust right-lateralized activation in the DLPFC, PPC, and FIC during “movement transitions” in the auditory event segmentation task. Here, we extend these findings to characterize network-specific responses in the CEN, DMN, and SN. Activations in the CEN and SN were found to be accompanied by robust deactivation in the DMN at the movement transition [Fig. 1A and General Linear Model Analysis in supporting information (SI Materials and Methods)]. To further confirm that these regions constitute coherent networks, rather than isolated regional responses, we performed independent component analysis (ICA) on the task data, which revealed the existence of statistically independent CEN, SN, and DMN (Fig. 1B, see also Table S1) [ICA is a model-free analysis technique that produces a set of spatially independent components and associated time courses for each subject (25)]. In the following two sections, we examine the putative causal mechanisms involved in switching between activation and deactivation in the context of the three networks, identified above, using a combination of mental chronometry and GCA (21, 22).

Latency Analysis Reveals Early Activation of the rFIC Relative to the CEN and DMN. First, we identified differences in the latency of the event-related fMRI responses across the entire brain using the method developed by Henson and colleagues (26). Briefly, this method provides a way to estimate the peak latency of the BOLD response at each voxel using the ratio of the derivative to canonical parameter estimates (see SI Materials and Methods for details). This analysis revealed that the event-related fMRI signal in the right FIC (rFIC) and ACC peaks earlier compared to the signal in the nodes of the CEN and DMN, indicating that the neural responses in the rFIC and ACC precede the CEN and DMN (see Fig. S1 and Table S2). To provide converging quantitative evidence, we estimated the onset latency of the blood oxygen level dependent (BOLD) response in these regions using the method of Sterzer and Kleinschmidt (27). Previous studies have used differences in the onset latency of the BOLD response as a measure of the difference in onset of the underlying neural activity (20, 21, 27). We first defined regions of interest (ROIs) in six key nodes of the SN, CEN, and DMN based on the peaks of the ICA clusters (see Materials and Methods); all subsequent analyses was confined to these six canonical nodes of the SN, CEN, and DMN (see also SI Text for a discussion on the choice of regions of interest and control analyses on regions not included in the main analysis). We extracted the mean time-course in each of these six nodes, and used a sixth-order Fourier model to fit the event related BOLD response for each subject and event, and averaged the fitted responses across events and subjects (see Fig. S2). Onset latencies were then computed as the time point at which the slope of the fitted response reached 10% of its maximum positive (or negative) slope in the initial ascending (or descending) segment. We found that the rFIC onsets significantly earlier than all of the nodes in the CEN and DMN (two-sample t-test, q < 0.05; FDR correction for multiple comparisons) (Fig. 2, see also Table S3). These results confirm that activity in the rFIC onsets earlier compared to the activation in the CEN nodes, and deactivation in the DMN nodes.

GCA Reveals that the rFIC Is a Causal Outflow Hub at the Junction of the CEN and DMN. Finally, to elucidate the dynamic interactions between the three networks we applied GCA. Briefly, GCA detects causal interactions between brain regions by assessing the