The relationship between evoked and induced EEG/MEG changes: Going beyond labels

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Researchers using EEG/MEG to study the link between brain activity and cognition have relied on one central tenant: the event-locked averaging of single trials of activity in the time-domain will result in the cancellation of any aspect of the signal not time-locked AND phase-locked to the event. However, while this assumption has led to a wealth of discoveries about how the brain processes information from the outside world, there is evidence that it's not necessarily correct. In the current chapter, I will go over the evidence that the ongoing electrophysiological signals detected at the scalp can never entirely be averaged out. Moreover, I will argue that this assumption has led to the labelling of the different types of event-related changes to the EEG/MEG which while making the interpretations of data easier can serve to limit the questions we can ask about how the brain functions.

"The moment you label something, you take a step- I mean, you can never go back again to seeing it unlabeled" Andy Warhol

For more than a century now researchers have been examining the electrical potentials and magnetic fields measured at the scalp to understand what is happening inside our brains when we are carrying out various cognitive tasks. The primary approach taken by researchers has been to characterize how the electro/magentoencephalogram (E/MEG) signal changes in response to a particular 'event', whether it be a button press or the onset of an auditory tone. These changes have historically been labelled as either *evoked or induced,* with each label making assumptions about the origins of the change. The rationale behind evoked activity is that the brain produces a new response as a consequence of processing the event. This response is both time-locked and phase-locked to the experimental event. Induced activity, on the other hand, assumes that the brain has ongoing brain activity (i.e activity that is always there) independent of any additive activity, and the event modulates this ongoing activity, in a time-locked, but not necessarily phase-locked manner. If I can have the reader take anything away from the upcoming chapter it is that these 'labels' while at times useful can paint an incomplete picture of what is going on in the brain during cognitive processing. I will further argue that for the field to move forward in gaining a richer understanding of the link between brain and cognition, we need to rethink how we label the different types of EEG responses.

Evoked and induced: the assumption and labels.

The evoked potential approach assumes a large component of the electrophysiological signals detected at the scalp is not related to the processing of the phenomena under investigation. Here averaging multiple trials (Figure 1 A and B) of EEG epochs centered around the experimental event is needed to extract the "event" related EEG signal, called the event-related potential ERP for EEG measurements, and event-related fields (ERF) for magnetoencephalography (MEG) measurements. The ERP/F reflects neural activity precisely *time* and *phase-locked* in response to an event. The peaks and troughs in the ERP waveform which often follow a stereotypical temporal pattern of positive and negative voltage deflections, are classified as 'components'. These components are theorized by researchers to map onto various task-relevant cognitive processes [1].

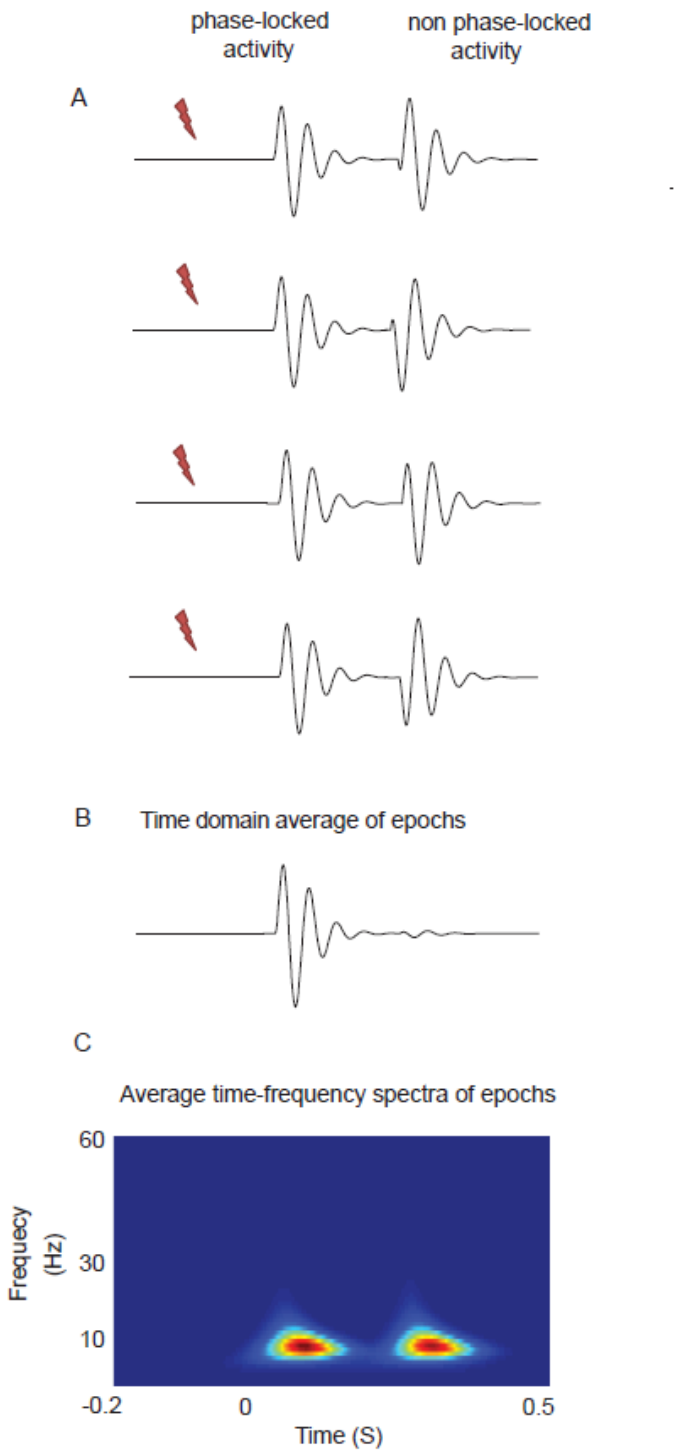

Figure 1 .A) The onset of an event (eg auditory stimulus) can **evoke** *activity that is both phase and time-locked to the onset of the event as well as* **induce** *activity that is time-locked but not phase-locked. B) Time-domain averaging of multiple data epochs would result in the attenuation of the non-phase locked activity due to destructive interference. The activity remaining after the averaging reflects the brain's transient phase-locked response to an event*

The evoked potential approach explicitly ignores the ongoing activity present in the EEG, as well as changes that although are time-locked to an experimental event, are not necessarily phase-locked to it. This is because of the rather critical assumption (and one which this chapter

will spend considerable effort arguing against) that non-phase locked activity disappears in the averaging of event locked data epochs due to the deconstructive interference of random phases(Figure 1B). Capturing changes to the ongoing activity in EEG, as well as responses that are time-locked but not necessarily phase-locked requires averaging the time-frequency spectra of multiple EEG trials centered on the experimental event (Figure 1C). The time-frequency characterization of the ongoing EEG activity works particularly well since the signals contain rhythms, that is oscillatory activity in characteristic frequency ranges (i.e. bands) including theta (3-7 Hz), alpha (8-13), beta (14-20 Hz), and gamma (30-100 Hz), with each band often exhibiting specific spatial distributions over the scalp [2]. The amplitude of an oscillation refers to the size of its (positive or negative) peak relative to some baseline.

The experimentally-driven increase in the amplitude of a frequency band is often referred to as an event-related synchronization (ERS). The ERS terminology is based on the fact that when the activity of neurons becomes synchronized, the spatial summation of the post-synaptic potentials results in an amplitude increase ([3]). Conversely, desynchronization of the neuronal population firing results in the cancellation of post-synaptic potentials, and as such, a drop in oscillatory amplitude within a frequency band, sometimes referred to as event-related desynchronization (ERD). Much like the ERP components, the task-related changes in oscillatory amplitudes have been found to map on to different facets of cognition [2, 4].

Going beyond Evoked and induced: the origin of the changes

Besides the methodologically different approaches needed to extract each type of change, evoked and induced activity are also thought to reflect different processes occurring in the brain in response to an outside event. One rather now-classic framework [3] proposes that evoked changes (i.e. ERPs) are assumed to occur because of event locked changes of afferent activity into cortical neurons. On the other hand changes in oscillatory power of the ongoing EEG are hypothesized to emerge due to the interaction of neurons and interneurons that control the frequency components of the ongoing activity.

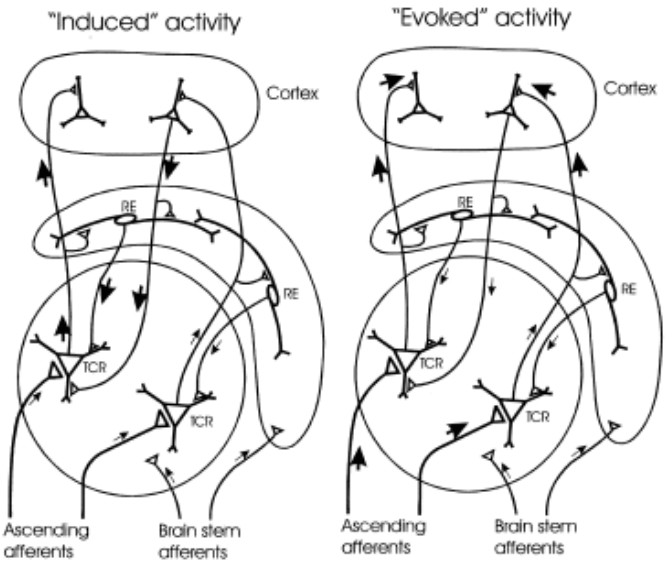

Figure 2 Schema for the generation of induced (ERD/ERS) and evoked (ERP) activity whereby the former is highly frequency-specific. Adapted from [3]

UNLabeling the labels: the relationship between evoked and ongoing activity

The rather traditional view of evoked and ongoing activity is that they reflect rather separate distinct neural phenomena. According to this view, the evoked activity which always has a consistent phase-locked to the onset of an experimental event rides on top of the ongoing activity. This is also sometimes referred to as the 'additive view' of how ERPs are generated. Taken to the extreme one could view the evoked activity is completely independent of the ongoing activity (figure 3A). An alternate theory, referred to as a phase-resetting theory (figure 3B), postulates that there is no additive evoked activity elicited by the onset of an event, but that rather, the ongoing activity adjusts its phase to the onset of the experimental event [5]. Here, by averaging trials locked to the event, the ongoing activity before the onset of the event which has random phases is averaged out, while the event-

related phase perturbed activity emerges as the evoked response

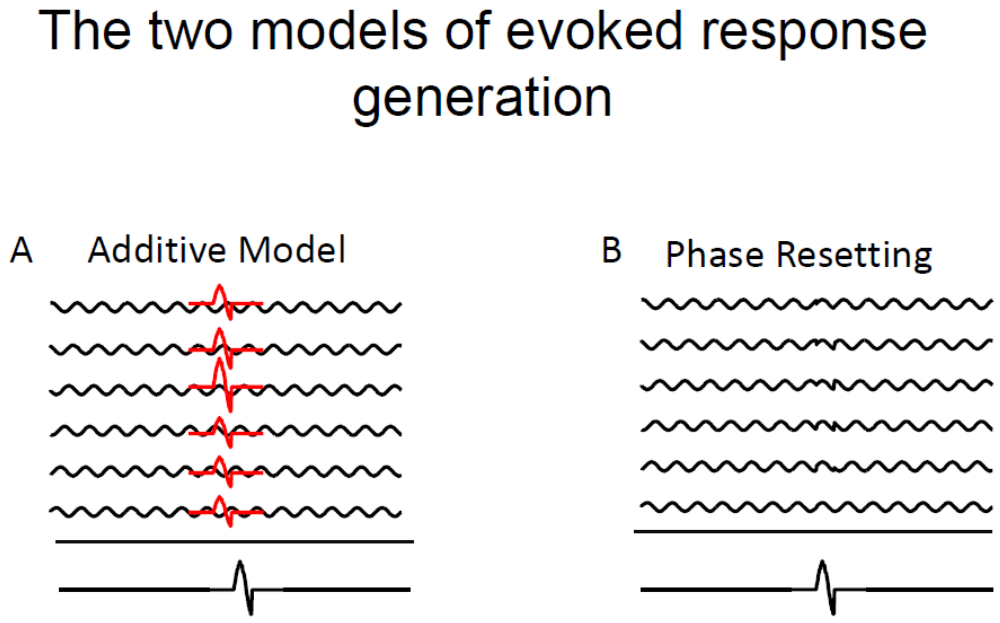

Figure 3. The additive versus phase-resetting theory of evoked response generation. (A) The additive theory assumes that evoked and ongoing activities are distinct neuronal phenomena. The experimental event "evokes" an additive, phase-locked response in each trial. (B) According to the phase-resetting view, the ongoing and evoked activity are the same neuronal phenomena, with no 'new' additive response. Here the phases of the ongoing background oscillations become aligned (phase-reset or partial phase-reset) to an experimental event. The phase-locked (i.e adjusted) oscillatory activity emerges as an evoked component when averaging the event-locked trials

Given that the predominant ongoing activity in the EEG signal is the alpha rhythm, it is believed that its phase-reset (or adjustment) to the onset of the experimental event plays a particular role in the formation of evoked responses [5] [6] [7] [8]. However, the phase-reset of the ongoing rhythms is not exclusive to the alpha activity, with the theta rhythm also proposed to be involved in the formation of specific evoked responses such as the error-related negativity([9]).

There has been a fair amount of controversy over whether phase-resetting can account for the formation of ERPs [10]. The primary evidence for the occurrence of a phase-reset is that the phase of the ongoing activity at the time of the evoked response would be consistent across trials. However, the addition of a signal with a consistent phase across trials (i.e. a

traditional additive evoked response) would also make the phase of the ongoing activity appear consistent across trials (figure 4) [11] [12] [13].

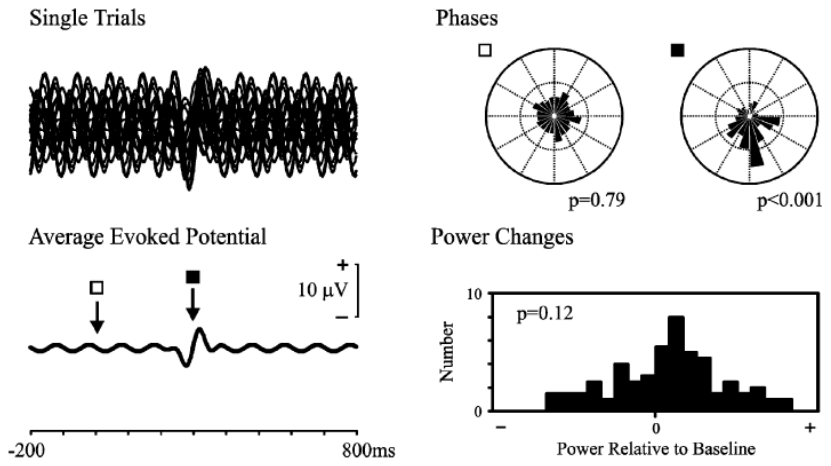

Figure 4 Model of generation. The upper left of the figure shows 20 superimposed trials of a model evoked potential consisting of a single cycle of activity added to ongoing activity of the same frequency with variable phase and amplitude. Below is given the average evoked potential over 100 trials. At the upper right are the polar plots showing the phase distributions of the frequency of the evoked potential (and background activity) during the baseline and at the middle of the evoked potential. There is significant phase synchronization at the time of the evoked potential. At the bottom is a histogram of the power measurements in the middle of the evoked potential across the 100 trials (because of the Mortlet filtering effect this gives the maximum power). There is no significant change in power. (reprinted by permission from [11]

Moreover, a rather convincing argument has been raised that the additive and phase-resetting model cannot be mathematically distinguished at the scalp level, without invasive electrophysiological recordings [14]

While the additive and phase-resetting theories offer a contradictory account of evoked and ongoing activity, they do share two common elements. Both theories assume across-trials averaging results in the attenuation of ongoing activity. However, as I will detail in the next section, this assumption has been challenged. There is now compelling evidence that the alpha rhythm, the dominant ongoing signal detected at the scalp, is non-sinusoidal, and across-trials averaging never really makes it go away. This observation greatly blurs the line between evoked and ongoing activity. Furthermore, it is important to note that the the additive and phase-resetting debate has exclusively focused on the early-stimulus evoked responses (P1, N1, or the ERN) and fails to provide a complete account for the brain responses occurring 200 ms after an experimental event. These sustained responses (figure 5) often lasting 100-200 ms are believed to reflect neural processing related to high-level cognitive constructs ranging from working memory representation [15] to language comprehension [16].

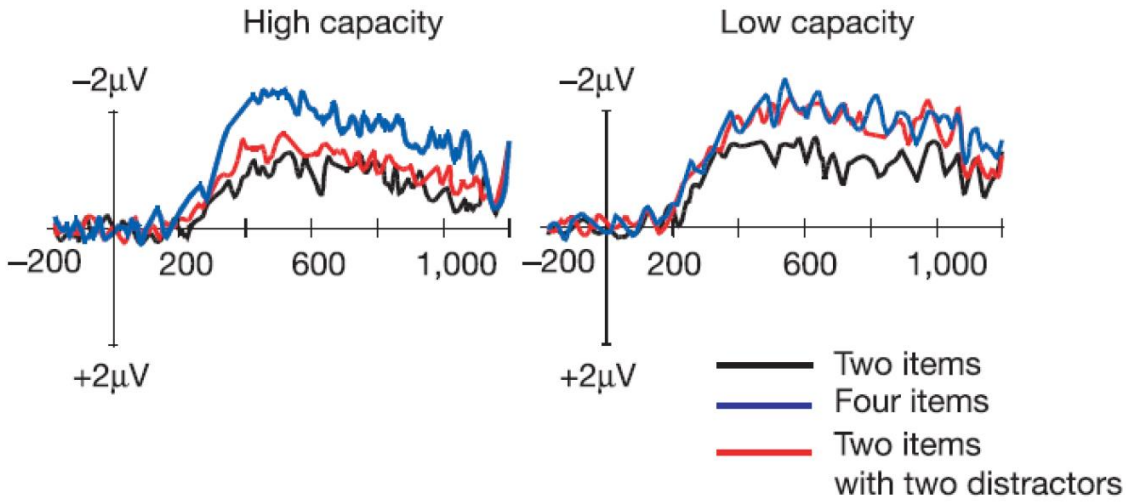

Figure 5 Grand averaged ERP difference waves (contralateral activity minus ipsilateral activity) timelocked to the memory array averaged across the lateral occipital and posterior parietal electrode sites and divided across the high and low memory capacity groups [17]

Amplitude asymmetry/baseline shifts- a unifying perspective ?

Ongoing activity has been assumed to average out because it has traditionally been viewed to be amplitude symmetric in nature, meaning its peaks and troughs modulate at the same rate (figure 6 A)

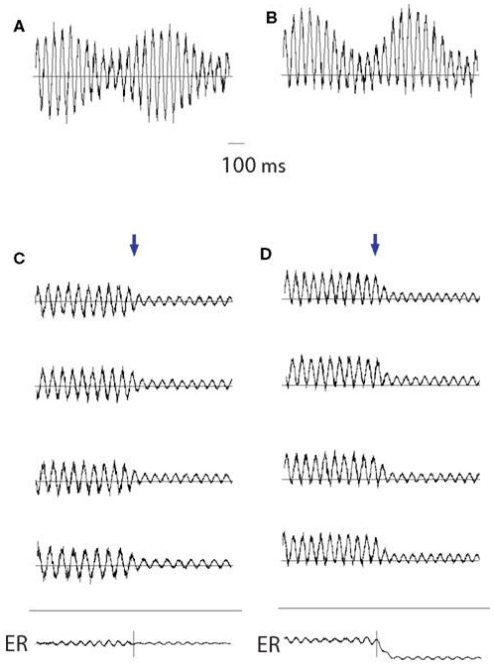

Figure 6 The amplitude modulation of neuronal oscillatory activity is conventionally viewed as being symmetric at approximately zero. B, We propose that the amplitude modulations of the oscillatory activity are asymmetric such that the peaks are more strongly modulated than the troughs. For the 10 Hz alpha activity, this could be explained by bouts of activity every ~100 ms. C, The conventional view ignoring asymmetric modulations of oscillatory activity would mean that averaging across trials (the arrow representing the start of the evoked response) would not result in the generation of slow fields. D, As a direct consequence of amplitude asymmetry, a depression (or increase) in alpha activity in response to a stimulus will result in the generation of slow fields when multiple trials are averaged

While a complete understanding of neural of origin of the scalp electrophysiological signals still remains to be elucidated [18] the general consensus is that they are generated through synchronized post-synaptic current in the dendrites of pyramidal cells [19]. Here the EEG

reflects the potentials by these currents, while MEG captures the magnetic fields. For an oscillation to have symmetric amplitude fluctuations the intracellular currents propagating forward towards the soma (here let us arbitrarily designate as the peak of the oscillation) must have the same magnitude as the current coming back from the soma (here assume the trough of the oscillation). However, given the asymmetric placement of channels responsible for the depolarization and repolarization current it is unlikely that the two currents would have the same magnitude when summed up across many synchronized neurons with the same orientation[20] [21].

An alternative way to view ongoing activity is that is it amplitude asymmetric, with greater variability in amplitude fluctuations at the peak versus the trough (figure 6 B). One critical consequence of amplitude asymmetric ongoing activity is that it will not simply average out to zero when summed across trials. Moreover, any systematic suppression or enhancement of the amplitude of the ongoing activity time-locked to an event would result in the amplitude envelope of the ongoing activity emerging as a slow evoked response when averaging across trials.

Empirical evidence supporting amplitude asymmetry

A seminal 2007 study by Nikulin et al [21] provided evidence that the ongoing alpha rhythm is 'amplitude asymmetric', specifically referred by them as having a 'zero-mean'. Nikulin et al went further to propose that a critical consequence of an amplitude asymmetric ongoing rhythm is that any systematic fluctuations in its amplitude would show up as slow responses (they referred to as baseline shifts) when averaged across trials (figure 7).

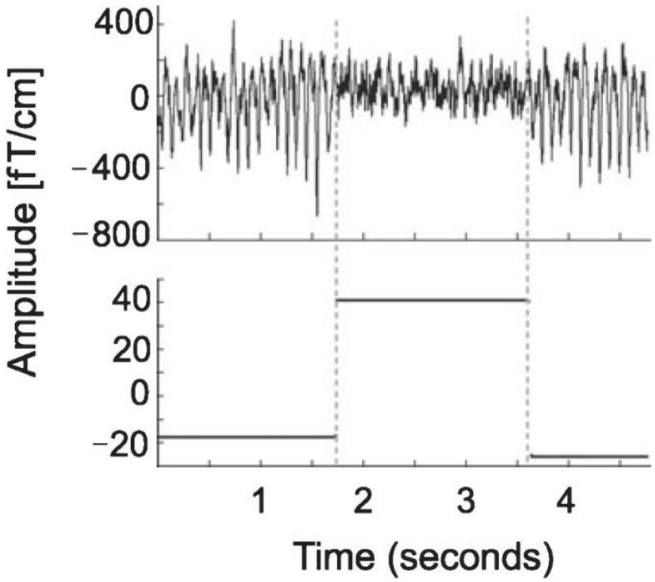

Figure 7 Baseline shifts in ongoing oscillations. (A) Upper trace: spatially filtered (with independent component analysis) broadband signal from a channel above the right sensorimotor area during rest; lower trace: the mean values in three time intervals. Clearly, there are baseline shifts in the ongoing activity associated with oscillations changing from large to small and back to large amplitude. If many epochs with similar amplitude dynamics are averaged, oscillatory patterns would disappear whereas the baseline shifts would remain leading to the appearance of an evoked response. [21]

Following up the work of [21], Mazaheri and Jensen (2008) developed a simple measure to quantify the amplitude of an oscillation by comparing the variance of its peaks with the variance of the troughs (see figure 8).

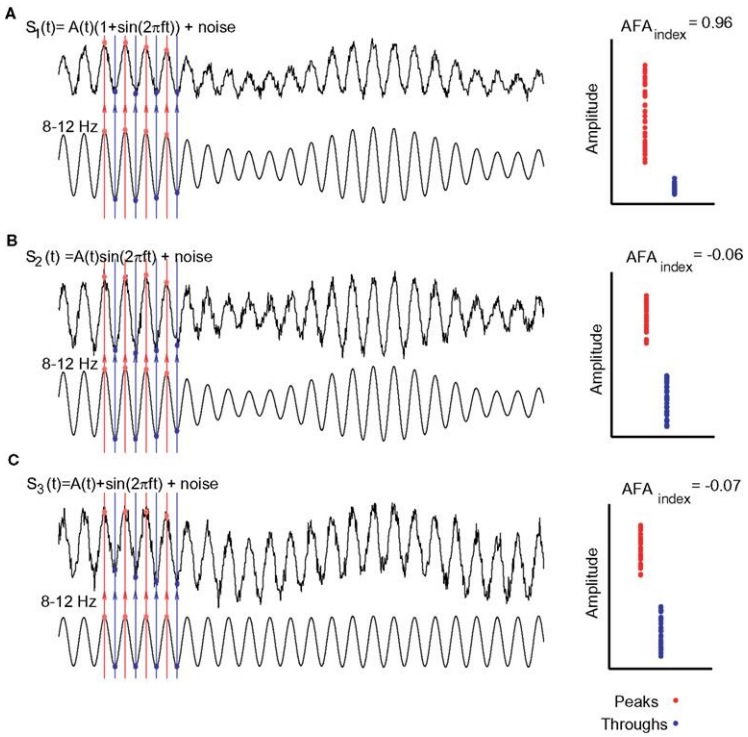

Figure 8. Various simulations in which surrogate signals were used to test the AFAindex. (A) The signal, s1(t), was designed to have an amplitude asymmetry. The amplitude modulation was determined by a slower signal A(t). Clearly the peaks (red dots) are more modulated than the troughs (blue dots) yielding a strong AFAindex. (B) The signal, s2(t), was designed such that the slow modulations, A(t), affected the alpha rhythm in a multiplicative manner. Thus peaks and troughs are modulated symmetrically over time yielding an AFAindex close to 0. (C) In signal s3(t) slow modulations were added to the alpha oscillations (DC-like offset of the signal). This affected peaks and troughs in the same direction producing an AFAindex close to 0. Adapted from [20]

Moreover, we were able to demonstrate that the degree of amplitude asymmetry of an oscillation was directly related to the amplitude of the evoked response generated by its modulation. Specifically, we presented a simple check-board stimulus across many trials, and then separated the trials into high amplitude of post-stimulus activity, and low amplitude. We found that despite the stimulus being the same, the sorting of the trials based on alpha amplitude resulted in the formation of slow-evoked responses (figure 9). Across participants the amplitude, and polarity of these slow responses was highly correlated with the direction of the amplitude asymmetry of the ongoing alpha activity. Thus we were able to demonstrate (albeit with simple grating stimuli) that it was in principle possible to form slow-evoked responses in the trial averaged EEG epochs if there were systematic changes in the amplitude of the ongoing alpha activity.

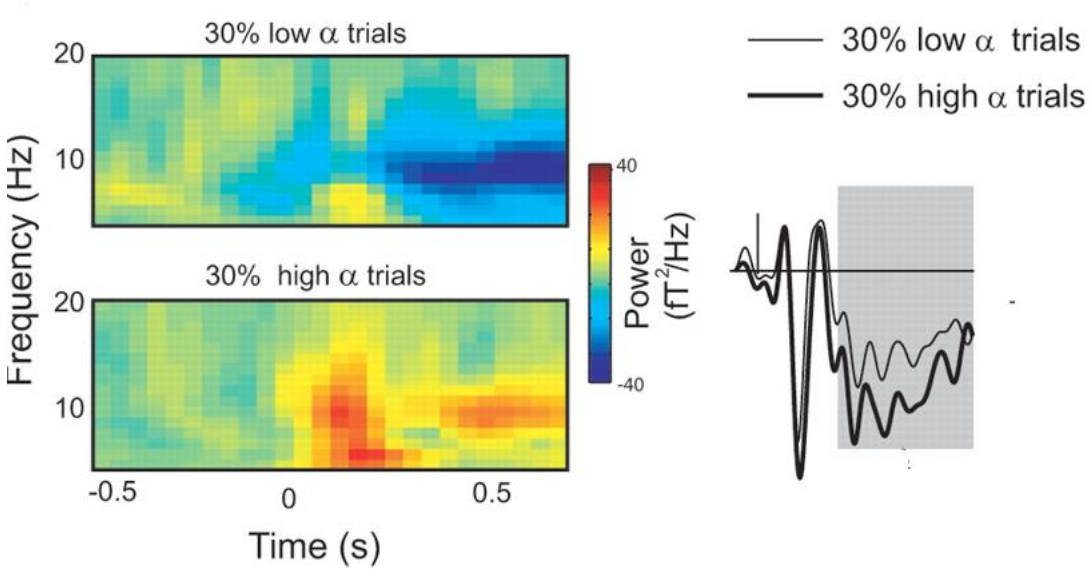

Figure 9 Time-frequency representations of the trials with the 30% lowest and 30% highest modulations of alpha power (TFRs baseline corrected; −0.6 < t < −0.1 s) in a representative subject. The respective ERFs (right) reveal a clear difference in the sustained modulation with respect to low- (thin line) and high-alpha-power changes (thick line). Adapted from [20]

In 2010 Mazaheri and Jensen [22] proposed four prerequisites for linking modulations of oscillatory activity to evoked component generation.

1. *The ongoing MEG/EEG oscillations must be modulated in amplitude by the stimuli or event*
2. *This amplitude modulation of the ongoing activity must correlate with the time course of the evoked response (over trials or subjects)*
3. *The ongoing oscillations must have an amplitude asymmetry*
4. *The magnitude and/or polarity of the amplitude asymmetry must relate to the amplitude and/or polarity of the evoked responses (over trials or subjects)*

Making the past as important as the future

One rather intriguing consequence of having ongoing activity that never averages out is that the amplitude of the pre-event oscillatory activity could modulate the amplitude of the post-event-related potentials, when baseline subtracting the event-related potentials (figure 10). A recent comprehensive study systematically examined the relationship of pre-stimulus power of oscillatory, amplitude asymmetry, and the formation of evoked responses [23]. In particular the authors here focused on differentiating the impact of pre-stimulus functional inhibition[24] (a sensory state being in a less-responsive state) from amplitude asymmetry on both the early and late sensory evoked responses.

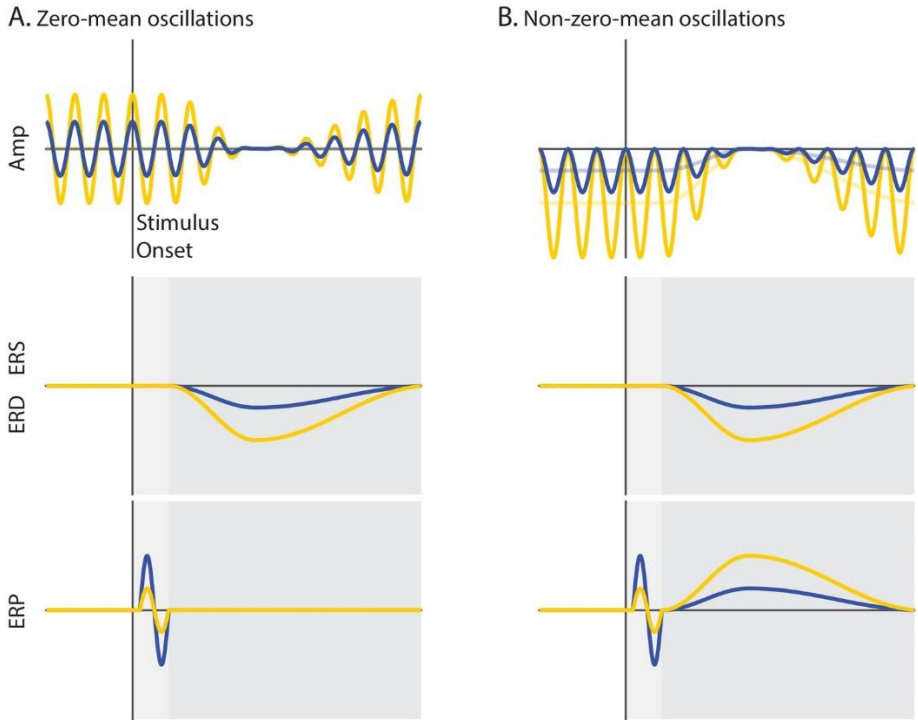

Figure 10 Ongoing activity (Amplitude), event-related oscillations (ERS/ERD) and potentials (ERP) are illustrated in upper, middle and lower panels, respectively. The vertical line indicates stimulus onset, while the horizontal line indicates zero signal strength. Yellow and blue represent states of strong and weak prestimulus power, respectively. (A) Non-phase-locked ongoing oscillatory activity with a zero-mean. The oscillations are symmetrical relative to the zero line of the signal (A upper panel). (B) Non-phase-locked ongoing oscillatory activity with a non-zero-mean. The oscillations are asymmetrical relative to the zero line of the signal. The signal baseline is characterized by a negative offset (opaque lines). The stronger the power of these oscillations, the stronger the negative offset of the signal baseline (B upper panel). During event-related desynchronization (ERD), the ongoing oscillations are suppressed to the zero line of the signal. This implies that the stronger the prestimulus power, the stronger the ERD (A/B middle panels). Trial averaging of zero-mean oscillations eliminates prestimulus oscillatory activity that is not time-locked to the stimulus because opposite oscillatory phases cancel out. This results in baseline signal at the zero line, which is unaffected by ERD. Therefore, an ERD of zero-mean oscillations does not generate the slow ERP component during the late time window because there is no baseline shift for these oscillations (dark gray; A lower panel). Trial averaging of non-zero-mean oscillations does not eliminate non-phase locked ongoing activity. This results in a prestimulus baseline signal with an offset relative to the zero line. During the ERD, the baseline of the signal gradually approaches the zero line of the signal. When the post-stimulus signal is corrected with the prestimulus non-zero baseline, a slow shift of the ERP signal appears, mirroring the ERD time-course. Specifically, an ERD of negative (positive) non-zero mean oscillations shifts the signal upward (downward), generating the slow ERP component of positive (negative) polarity. Crucially, the stronger the prestimulus power, the stronger the ERD, and as a consequence, the stronger the slow shift of the ERP. The baseline-shift account predicts a positive relationship between prestimulus power and the amplitude of the slow ERP during the late time window (dark gray; B lower panel). According to the functional inhibition account, strong prestimulus power attenuates the amplitude of the additive ERP components. This account predicts a negative relationship between prestimulus power and the amplitude of ERP components during the early time window (light gray; A/B lower panels).

Here with functional inhibition the authors were referring to the currently widely held view that an increase in alpha activity in a sensory system reflects its functional inhibition and consequently results in attenuated evoked responses (evidence recently reviewed in [24]). They found that the early evoked (<0.200 s: eg the C1/N1 components) were indeed modulated by the amplitude of the pre-stimulus alpha activity, independent of the direction of amplitude asymmetry. However, they found a strong relationship between amplitude asymmetry of the pre-stimulus activity and the late evoked components These results taken together suggest high pre-stimulus alpha likely causes a suppression of early evoked

responses since the neurons producing these responses are in an inhibited stated, while the amplitude of asymmetry property of alpha activity impacts the formation of the later slow evoked responses.

The results I have discussed so far strongly question the old dogma that ongoing and evoked activity are distinct independent neural phenomena. However, while the studies demonstrate that it is possible to generate slow evoked potentials through modulations of the amplitude of the ongoing activity (without any 'new' additive activity), it is still unclear if this mechanism applies to cognitively relevant event-related responses.

Can *amplitude asymmetry explain the emergence of the most cognitive of ERPs?*

As mentioned earlier, the CDA is a slow sustained response that is proposed to reflect the neural representation of an item in working memory. It is often elicited through a paradigm where participants are presented with a bilateral array of colored squares and instructed to memorize the location of the items in the hemifield indicated by the arrow (i.e., test array). The success of memorizing the items in the test array is then subsequently assessed a second later through the presentation of another array that is either identical to the test array, or missing one of the items. The CDA is derived by averaging epochs locked to the onset of the test array and subtracting the contra-lateral ERPs from the left.

The amplitude of the CDA is modulated by the number of items held in working memory[17] However, the neural origins of the CDA are still rather a mystery. Moreover, the same paradigm has also been found to elicit robust modulations of alpha activity that are also modulated by the number of items held in working memory [25]. In addition, just like the CDA the degree of lateralized alpha modulation also seems to correlate with the individual differences in working memory. This suggests some overlap between the neural processes underlying the CDA and the alpha modulation.

A study published by Van Dijk (2010) et al [26] explored the link between changes in alpha activity and the CDA, and found them to be quite linked together. Specifically, they observed that both the degree of alpha suppression across individuals, as well as their degree of alpha amplitude asymmetry correlated very strongly with the amplitude of the CDA. Moreover, the alpha modulation and the CDA had a remarkably similar topography over the scalp. These observations taken together could suggest that the CDA and the alpha modulation during the period that the items are held in working memory are one and the same thing.

What are the consequences of re-labelling the CDA as a change in ongoing activity rather than a purely additive response? For one thing this could have profound implications on how we believe the brain carries out working memory processes. As mentioned earlier one popular view of the role of alpha modulation in cognition is the suppression of task-irrelevant regions [24]. Thus, the CDA rather than being an additive neural process involved in memory maintenance could be reflecting the inhibition of task-irrelevant brain areas. Additionally, unifying ongoing and event-related activity has the potential to mechanistically account for some rather intriguing ERP findings, for which the origins of the responses remain a mystery. For example, a now classic study[27] found that the amplitude of slow event-related potentials locked to the onset of a cue but peaking before the onset of a word to be remembered, could predict if the word was later remembered. By linking the slow ERPs to the modulation of ongoing alpha activity, one simple interpretation of the observed difference between the

Remembered versus Forgotten words could be that alpha activity is higher(i.e the brain is in a more inhibited state) prior to the onset of forgotten words. This is indeed in line with several experiments observing pre-stimulus alpha oscillations to modulate perception [28] as well as reflect slips of sustained attention [29].

DOES *amplitude asymmetry explain the emergence of most cognitive of ERPs?*

While I hope that I was able to so far demonstrate that modulations of ongoing activity that is amplitude asymmetric can produce sustained ERPs, the jury is still out on whether the ongoing and evoked activity, particularly the slow late components are one and the same. One study that has directly challenged this view is the observation that while alpha modulation and CDA are tightly linked, they do appear to uniquely contribute to individual differences between working memory capacity [30]. Specifically, the authors reasoned if the alpha suppression and CDA are two sides of the same neural phenomena they should also show the same relationship to individual differences in working memory performance. However, they found that each signal appeared to uniquely contribute to individual differences in working memory capacity.

A more recent study went further and used a decoding approach to investigate the specific roles alpha modulation and the slow sustained response could play in attention and working memory [31]. The authors found modulations in the ongoing alpha activity to be associated with the spatial location of attended stimuli, whereas the amplitude and spatial distribution of the slow-sustained ERPs were sensitive to orientation. Interestingly, the authors proposed that the ERP and alpha modulation, while serving distinct roles, reflect attentional mechanisms that prevent interference, rather than the actual WM representation.

While the studies just mentioned certainly do not rule out that the modulation of ongoing activity could be a significant contributor to the formation evoked responses, they do suggest the presence of additive activity involved in WM maintenance. In addition, the mechanism underlying amplitude asymmetry of alpha activity is also applicable to other frequency bands. This means that while the alpha rhythm is the predominant oscillation making up the ongoing activity, there could also be other rhythms present such as the delta and theta rhythms [32] whose event-related modulation likely impacts the formation of evoked responses.

Final thoughts

I would certainly not advocate any researchers to dismiss the event-related averaging approach in exchange for looking at changes in the brain's ongoing activity. However, strictly viewing ongoing activity and evoked responses as separate unique entities is implicitly believing the brain was not doing anything before the onset of the experimental event. Such a view is particularly limited when it comes to trying to understand how the brain tries to make sense of the outside world.

Just as an example, one rather influential theory on how the brain endeavors to make sense of the world proposes that the brain is constantly making predictions about what is going to happen next (reviewed in detail in [33]). Specifically, this theory, referred to as 'predictive coding', postulates that brain sets expectations and predictions about upcoming sensory input

and then subsequently updates these expectations after the onset of the sensory input. Here the discrepancy between the expectation and actual sensory input is referred to as prediction error. While evoked responses can reveal information about the degree of prediction error and the perceived mismatch between expectation and reality, they are not directly informative about the neurophysiology of the predictive processes themselves since by definition the evoked response emerges after the sensory input. By removing the separate labels (going back to the Warhol quote that started this chapter) of evoked responses and ongoing activity one can get a richer, but at the same time more parsimonious picture of the neural processes underlying cognition.

Finally, to close this chapter, I will paraphrase Warhol for one last time, I hope some of the mystery behind event-related responses and ongoing activity *is* gone but the amazement *is* just starting."

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