CHAPTER 4

Understanding the behaviors of E-DN

The reticular formation/system is responsible for 'alert' or 'wakeful' component of consciousness (medical definition). Within the dipole network (DN), tonic arousal is represented by B-stimulus (bias). This means that in a realistic scenario the DN does not receive continuous tonic arousal (B-stimulus) either due to inhibitions preventing arousal or due to exhaustion of neurotransmitters (metabotropic) responsible for arousal (Fig. 4.1).

This chapter deals with the hypothetical case of a single DN receiving continuous tonic arousal (B-stimulus). This is simulated with continuous run of B – D&B – B stimulus trial for number of times. The test run is done in various ways. For instance, duration of the dual-stimuli (D&B) packets is lengthened (from 1.5 to 2 seconds) for the simulation run shown in Figure 4.2.

At least two significantly different DN behaviors (with respect to Eck5 & Eck6 outputs) are observed. From the end of DN channel (Eck6) receiving just B-stimulus, one behavior is the rebound property during B-stimulus following dual-stimuli. Another is continuous spiking from Eck6 in contrast to the short spiking interval for rebound property. This later behavior is a departure from DN behavior (rebound) described in earlier chapter. It should be mentioned here that this is not the case for G-DN, whose behavior remains unchanged. The next step is therefore, investigation for the causes of such behaviors in the E-DN (and also the E-N) to help us better understand the network.

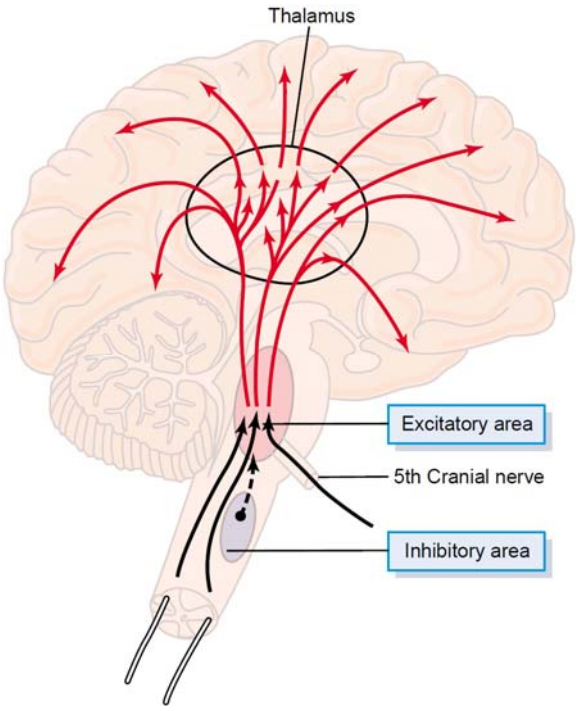

Figure 4.1. Reticular system of the human brain (adapted from [Guyton & Hall 2006]).

Reticular Excitatory area:

Location- This central driving component of reticular system is located in the reticular substance of pons and mesencephalon. This area is also called bulboreticular facilitory area.

Function (Peripheral)- Transmits facilitory signals down the spinal cord to maintain tone in antigravity muscles and control levels of reflex activities.

Function (Central)- Upward signals passing through thalamus can functionally be divided as: 1) fast action-potentials exciting cerebrum for few milliseconds (acetylcholine is the common neurotransmitter), and

2) slow signals (via small slow conducting fibers mainly in intralaminal & reticular nuclei of thalamus) build up progressively for seconds to a minute or more to control long-term background excitability level of the brain.

Reticular Inhibitory area:

Location- medial and ventral regions in medulla.

Function- Inhibit or depress the excitatory (or activation) system. One mechanism on how it does this is by exciting serotonergic neurons secreting inhibitory neurohormone serotonin at specific brain sites. For instance, serotonin released in diencephalons and cerebrum plays an essential inhibitory role to cause sleep.

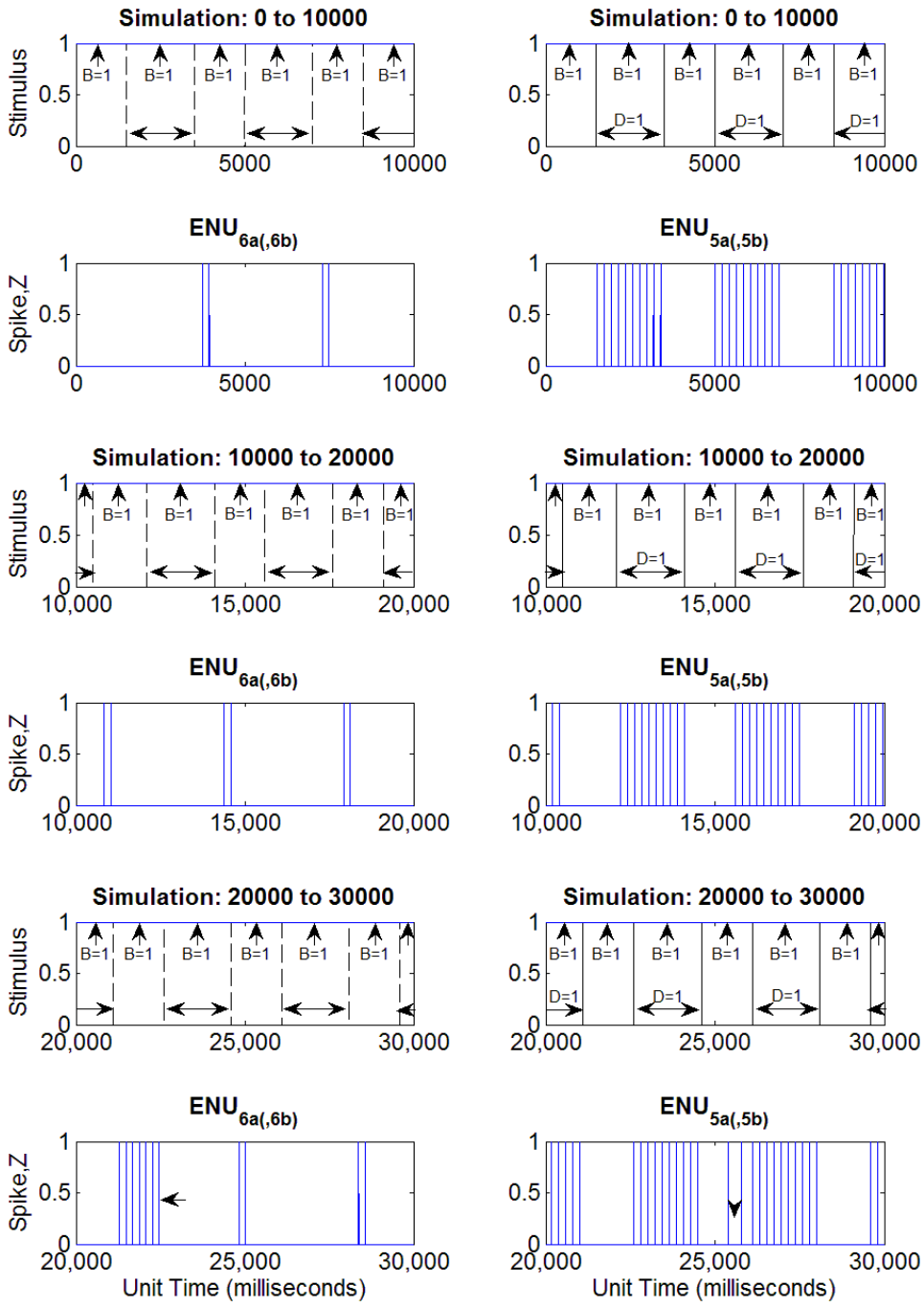

Figure 4.2. Simulation of E-DN part of E-N (Fig. 3.39) with recurring trial of B – D&B – B stimulus, such that duration of B-stimulus is 1500 ms and duration of dual-stimuli packets is 2000 ms. Above shows the simulation for 30 seconds. Arrow (bottom left corner) indicate node-6 (Eck6) spiking continuously during B-stimulus following dual-stimuli. Arrow-head (bottom right) indicate Eck5 spiking prior to dual-stimuli.

Basic ENU's within $ENU_{1m\text{-}elas}$ group at high-pass mode spiking relative to those in $ENU_{2m\text{-}elas}$ (all-pass mode) determines rebound property.

As mentioned earlier $ENU_{1m\text{-}elas}$ group is at the initial node (Eck1) getting inhibition from ENU_{1modu} triggered by dual-stimuli (extinguishing any activity from $ENU_{1m\text{-}elas}$ group). This inhibition forces $ENU_{1m\text{-}elas}$ to fire in high-pass mode while $ENU_{2m\text{-}elas}$ remains in all-pass mode. Thus, during B-stimulus following dual-stimuli $ENU_{1m\text{-}elas}$, now in high-pass mode, fires with a delay. During this delay $ENU_{2m\text{-}elas}$ activated ENU_4 sneaks some excitatory spikes into ENU_6 (but no inhibitory spikes from ENU_5). This triggers spiking from ENU_6. However, whether this ENU_6 spiking demonstrates rebound property depends on the relative spike occurrences between $ENU_{1m\text{-}elas}$ and $ENU_{2m\text{-}elas}$, post dual-stimuli. Note that spiking from either $ENU_{1m\text{-}elas}$ or $ENU_{2m\text{-}elas}$ has the same inter-spike intervals and the resulting spikes from ENU_3 and ENU_4 (both in all-pass mode) respectively are the reflections of their sources.

Figures 4.3 and 4.4 shows that if two ENU_3 spikes (double arrow, Fig. 4.4) activated by $ENU_{1m\text{-}elas}$ (in high-pass mode) after the delay occurs such that ENU_4 spikes (single arrow, Fig. 4.4) within a certain interval between the two ENU_3 spikes, then the ENU_4 spike is unsuccessful in exciting ENU_6 (in Eck6). This is also the case for all succeeding ENU_4 spikes. Thus, ENU_6 spikes during the delay demonstrate rebound property of the network. However, if ENU_4 spiking (single arrow, Fig. 4.6) occurs outside this interval Eck6 spiking ensues. That is, spiking from Eck6 occurs during the whole duration of B-stimulus following dual-stimuli (Fig. 4.5). This does not represent rebound property. We shall call this persistent-Eck6 spiking. Following these observations, next step would be the determination of interval during which ENU6 (within Eck6) spiking occurs.

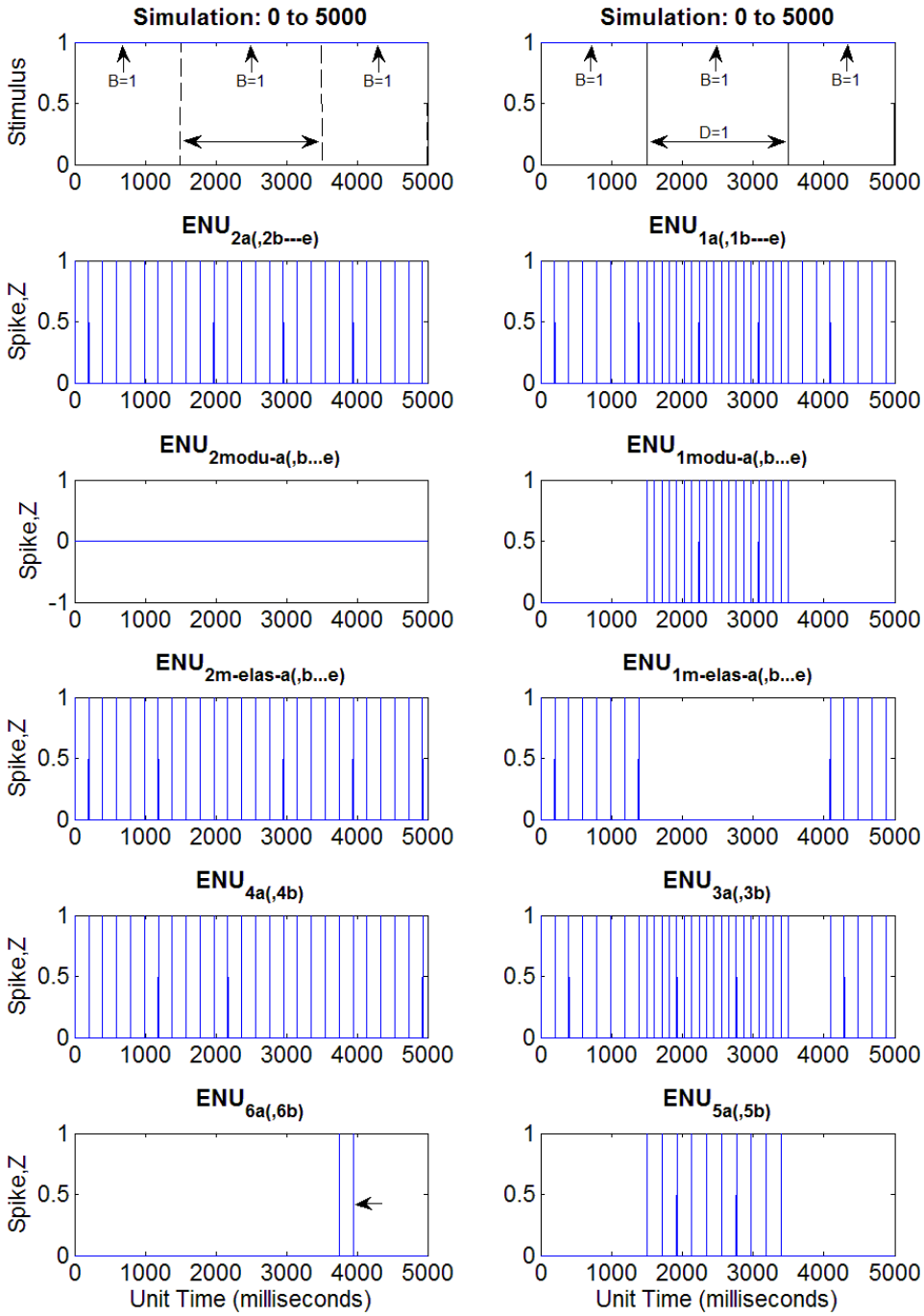

Figure 4.3. First 5 seconds of the simulation shown in Figure 4.2. This shows spiking within each node of E-DN (pulsed-inputs are not shown). During this B – D&B – B trial, the network exhibits rebound property (arrow, bottom left).

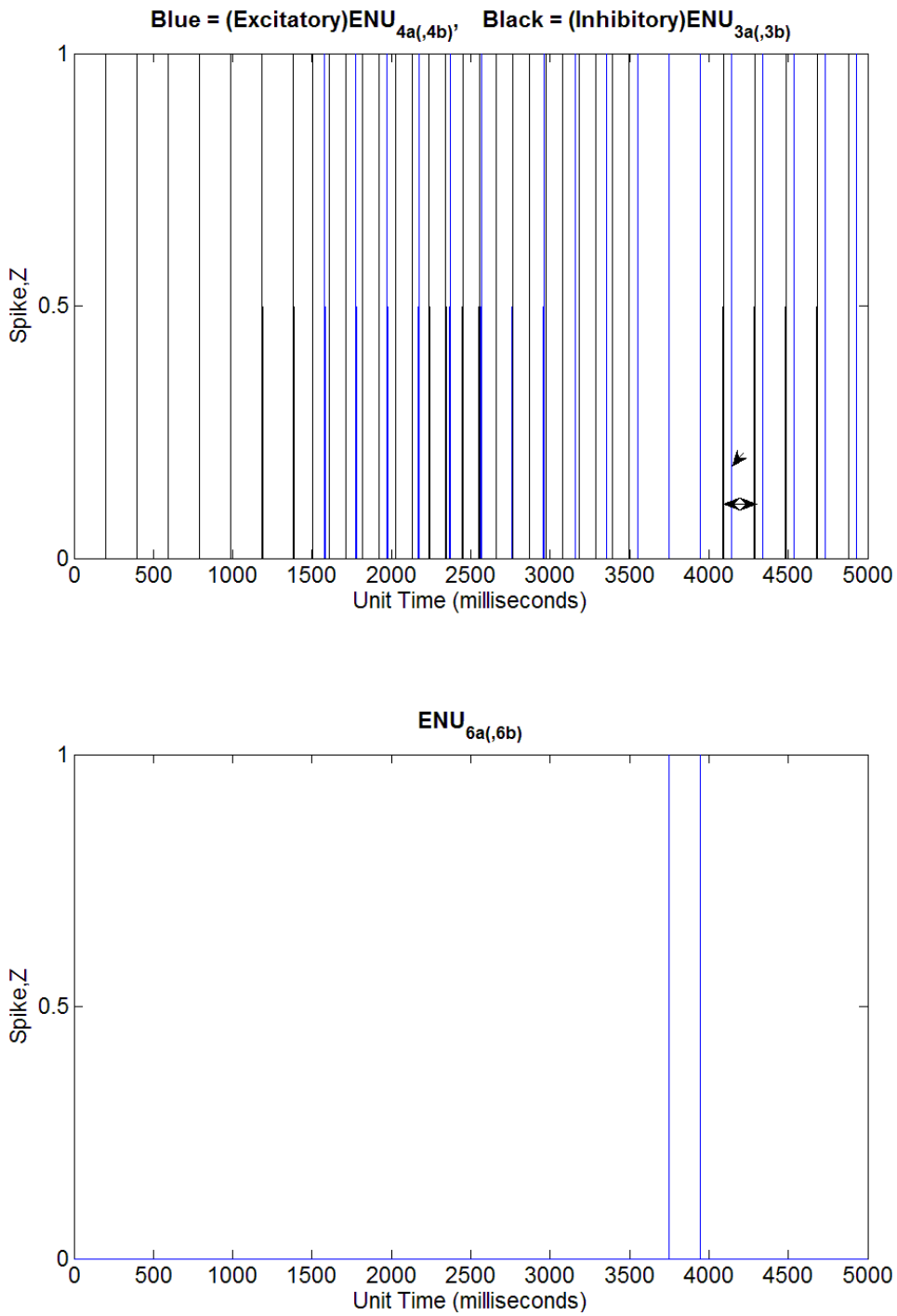

Figure 4.4. (Top) Inputs (spikes) for excitatory (blue) and inhibitory (black) dendrites of ENU_6 (basic ENU in Eck6) as seen in Figure 4.3. When excitatory input (arrow) occur outside a certain region between two inhibitory inputs (double-arrow), Eck6 spiking is not induced (bottom). Note that prior to 1500 ms both inputs (excitatory & inhibitory) occur at same instant.

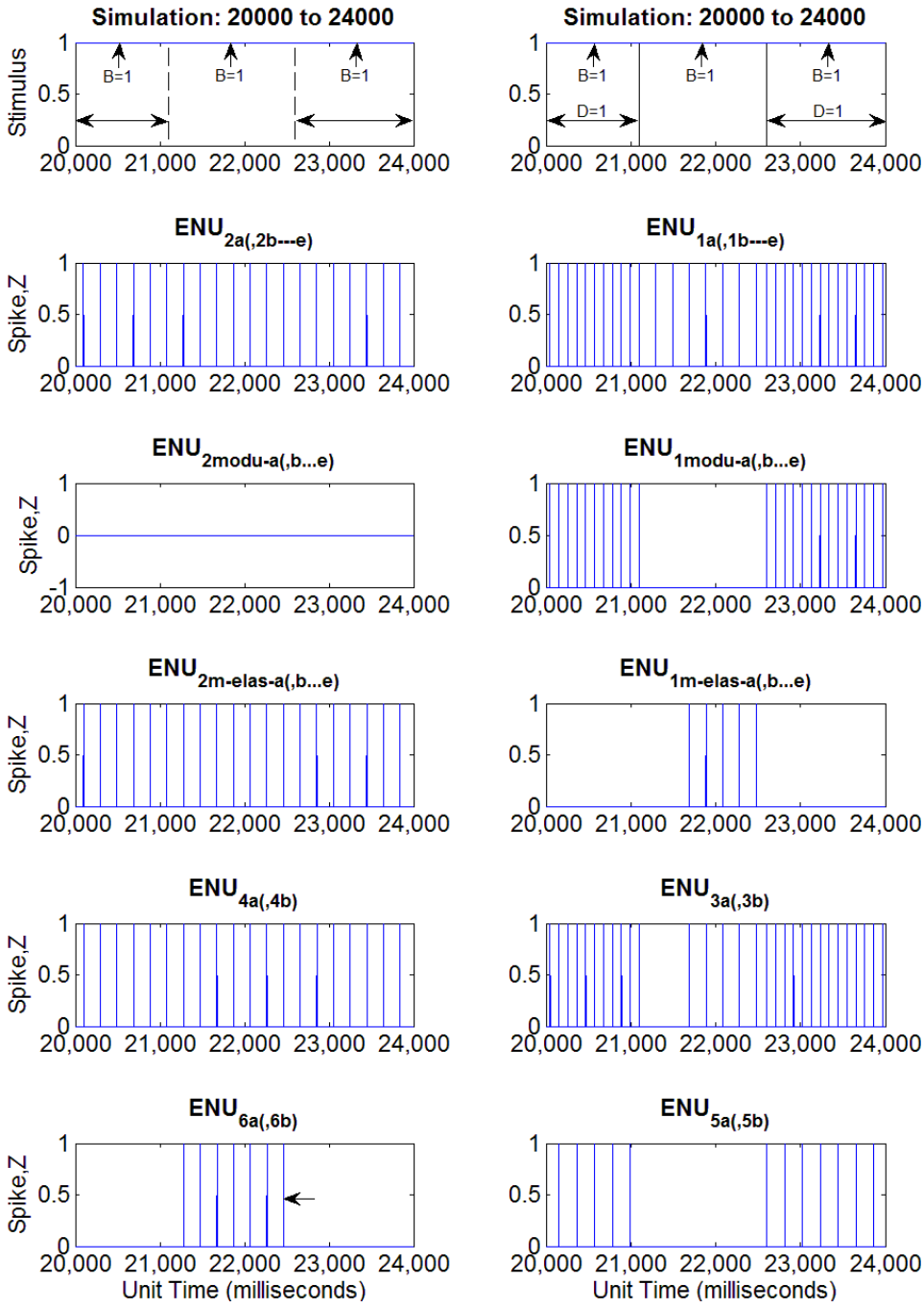

Figure 4.5. Spiking within each node of E-DN during simulation shown in Figure 4.2 (bottom). During this B – D&B – B trial (first B not shown), continuous spiking (arrow, bottom left) from ENU_6 (basic ENU in Eck6) occurs during the whole duration of B-stimulus following dual-stimuli.

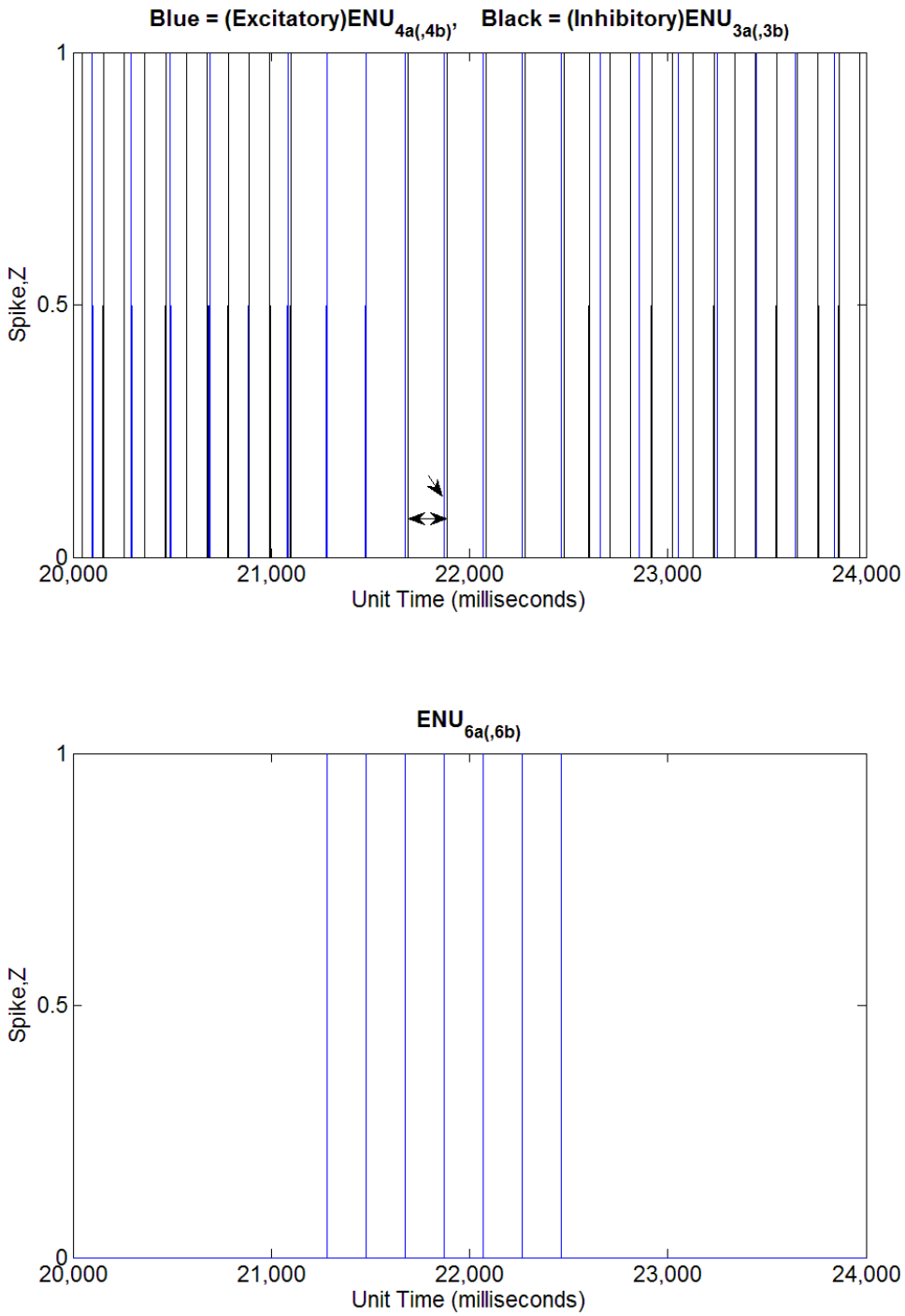

Figure 4.6. (Top) Inputs (spikes) for excitatory (blue) and inhibitory (black) dendrites of ENU_6 (basic ENU in Eck6) as seen in Figure 4.5. When excitatory input (arrow) occur within a certain region between two inhibitory inputs (double-arrow), Eck6 spiking is induced (bottom).

Instant of excitatory dendrite (feeding field) output and inhibitory dendrite output is crucial to soma output

The activated ENU_3 and ENU_4 spikes occurring at same instant of respective $ENU_{1m\text{-}elas}$ and $ENU_{2m\text{-}elas}$ spikes (Fig. 4.3 & 4.5) are the inputs for inhibitory and excitatory ENU_6 dendrites, respectively, and for excitatory and inhibitory ENU_5 dendrites, respectively. Therefore, the interval within which ENU_4 spikes can cause ENU_6 spiking is the interval during which excitatory dendrite output (from an ENU_4 spike) can successfully induce ENU_6 spiking (similarly for ENU_5 spikes but with different/reversed dendrite inputs). The analysis is done by considering ENU_6 (in Eck6) spikes.

For the dendrite components of basic ENU's within Eck6 we can calculate the maximum possible excitatory dendrite output (E_{ff}^{Max}) given by,

$$E_{ff}^{Max} = (1/\tau_{ff}^{E}) \cdot \sum_{i=1}^{n} w_{ff_i}^{E} \cdot (1 + (1/\tau_{lf}^{E}) \cdot \sum_{j=1}^{m} w_{lf_j}^{E}) \qquad (1)$$

where n = number of basic ENU's in either Eck3 or Eck4 and m = l - 1, l = number of basic ENU's in either Eck5 or Eck6. Note that the first component of the equation is of the form $\sum_{i=1}^{n} w_{ff_i}^{E} \cdot Z_i^{E}$. But for E_{ff}^{Max}, $Z_i^{E} = 1$ for all basic ENU's (i.e., for all i's). Thus,

$$\sum_{i=1}^{n} w_{ff_i}^{E} \cdot Z_i^{E} = \sum_{i=1}^{n} w_{ff_i}^{E} .$$

However, the linking field (LF) configuration implemented in E-DN and E-N (Fig. 3.34, 3.35 & 3.39) is such that the LF-component receives inputs from outputs (Z's) of basic ENU's other than itself within its respective ENU group. Thus, if an E_{ff} value is successful in causing a spike, a second peak in E_{ff} occurs (given by Eqn.1) as shown in

Figure 4.7.

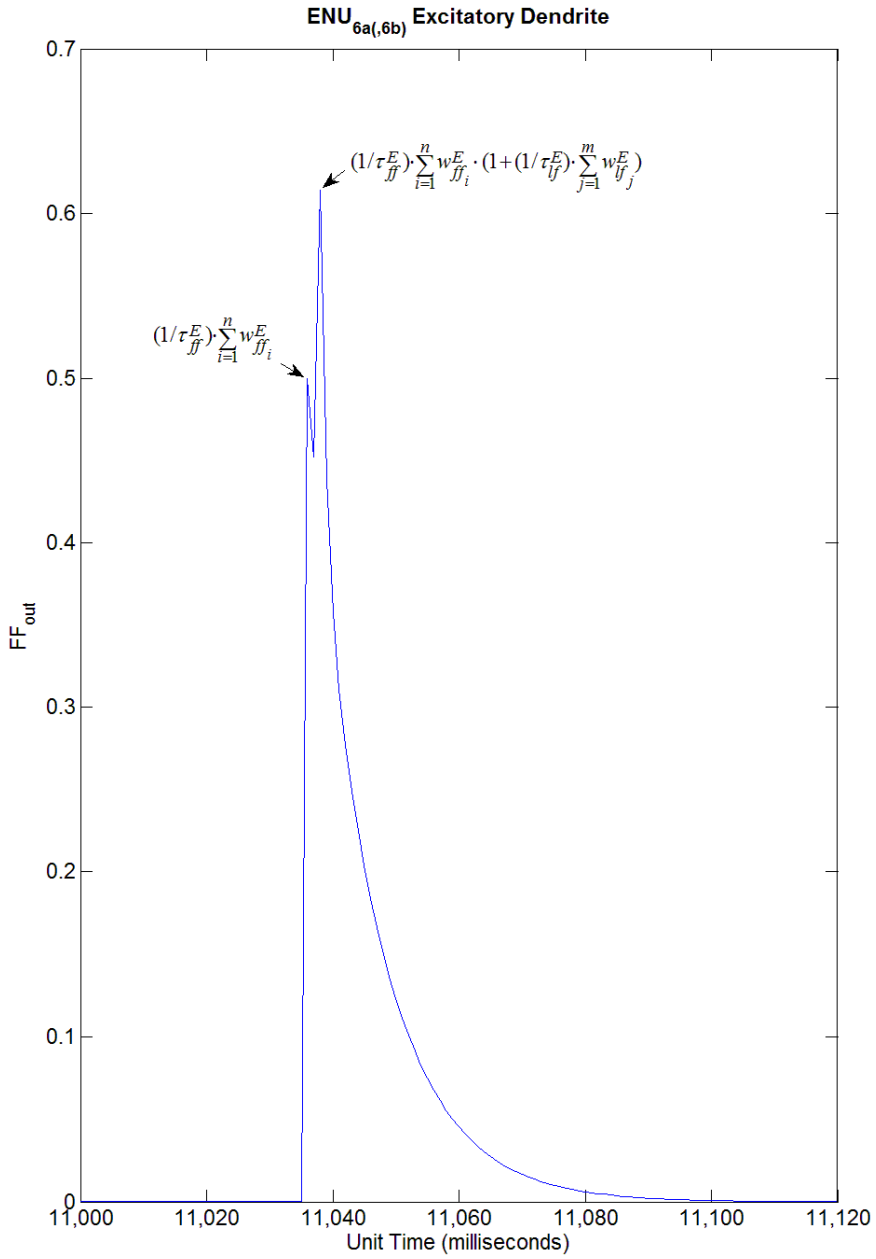

Figure 4.7. Excitatory dendrite output with two peaks. The first peak, if successful in inducing a spike from the soma, results in activating the linking field component of the excitatory dendrite. This is due to the network connection implemented in the network (Fig. 3.34, 3.35 & 3.39). Thus, second peak does not occur if the first does not succeed in causing spike from soma. The causes for the peaks are given by their respective equations. Note that inhibitory dendrites do not have any linking field connection and hence inhibitory dendrite outputs do not exhibit two peaks.

Because of the above reasons and since our interest lies in the first E_{ff} peak that determines spiking, we shall call this E_{ff}, the E_{ff}^{Max} given by

$$E_{ff}^{Max} = (1/\tau_{ff}^{E}) \cdot \sum_{i=1}^{n} w_{ff_i}^{E} \ . \tag{2}$$

Similarly, the maximum possible inhibitory dendrite output (I_{ff}^{Max}) is given by

$$I_{ff}^{Max} = (1/\tau_{ff}^{I}) \sum_{i=1}^{n} w_{ff_i}^{I} \ . \tag{3}$$

In addition to E_{ff}^{Max} and I_{ff}^{Max}, since value of the offset threshold parameter is also known (θ_o from table 3.10), the crucial instant (t_k) is when the inhibitory dendrite output is $I_{ff} = E_{ff}^{Max} - \theta_o$. Thus, if t = 0 and $\Delta t = t_k$, the difference equation for inhibitory dendrite output

$$FF^{I}(t) \ = \ FF^{I}(t-\Delta t) \cdot \exp(-\Delta t / \tau_{ff}^{I}) + (1/\tau_{ff}^{I}) \cdot w_{ff}^{I} \cdot \sum_{\forall j} F_j^{Inhibitory\ inputs} \tag{4}$$

can be re-written as,

$$I_{ff} \ = \ I_{ff}^{Max} \cdot \exp(-t_k / \tau_{ff}^{I}) \tag{5}$$

since, $FF_i(t_k) = I_{ff}$, $FF_i(0) = I_{ff}^{Max}$ and the dendrite does not receive any inputs. Therefore, taking the time when I_{ff}^{Max} precedes E_{ff}^{Max} as the reference (origin, t_o), if t_{spike} is the time from t_o when E_{ff}^{Max} (following I_{ff}^{Max}) occurs, t_k is the crucial time from t_o, and $t_{inter\text{-}inhibitory}$ is the time from t_o to the instant of next I_{ff}^{Max}, then for spiking to occur E_{ff}^{Max} should occur within the interval, $t_k \le t_{spike} < t_{inter\text{-}inhibitory}$. Hence, E_{ff}^{Max} must occur outside this interval for rebound (Fig. 4.8), i.e., no Eck6 spikes after the initial few spikes. On the other hand, E_{ff}^{Max} satisfies the condition (inside the interval) for persistent-Eck6 spiking (Fig. 4.9).

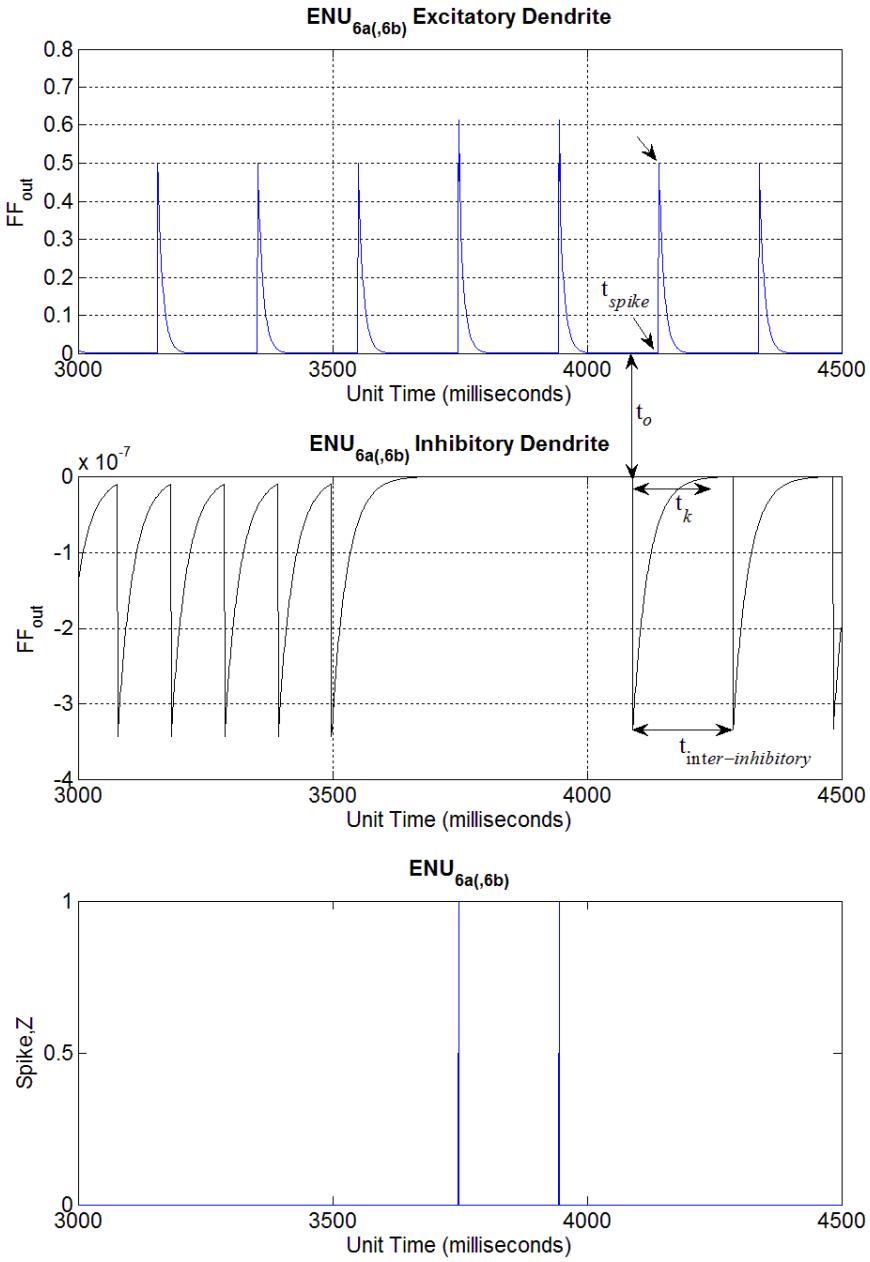

Figure 4.8. Eck6 spiking (rebound) seen in Figures 4.3 (bottom left) and 4.4 is because excitatory dendrite outputs (top, arrow) of ENU_6 during B-stimulus following dual-stimuli does not satisfy the condition, $t_k \leq t_{spike} < t_{inter\text{-}inhibitory}$ after the initial few spikes (which satisfied the condition).

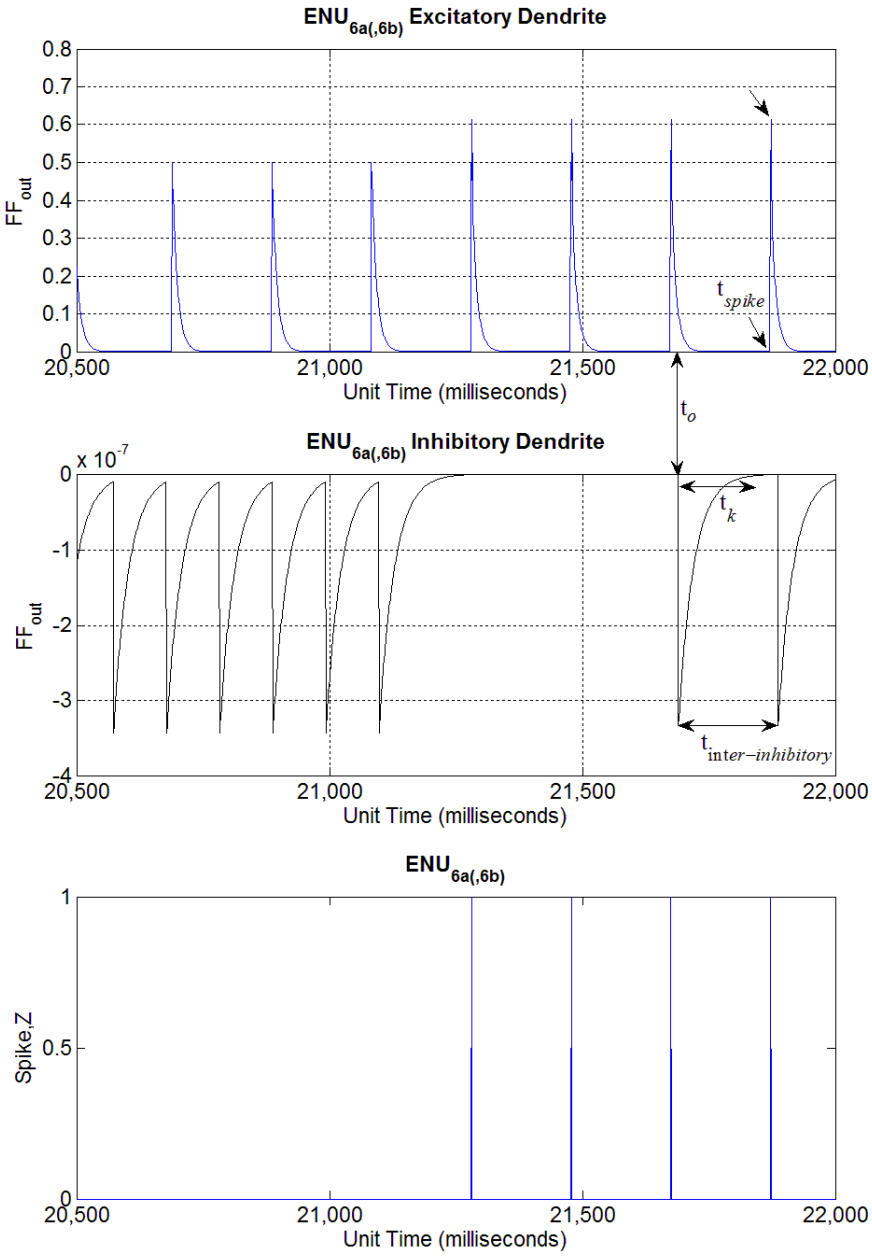

Figure 4.9. Eck6 spiking (persistent-Eck6 spiking) seen in Figures 4.5 (bottom left) and 4.6 is because excitatory dendrite outputs (top, arrow) of ENU_6 during B-stimulus following dual-stimuli satisfies the condition, $t_k \leq t_{spike} < t_{inter\text{-}inhibitory}$.

Instant of stimulus (switching 'on' or 'off') influences network behavior (Eck5 & Eck6 spiking)

Apart from the rebound property and persistent-Eck6 spiking, other network behaviors are also found. Occasionally, spiking from Eck5 occurs during B-stimulus following dual-stimulus (Fig. 4.2 bottom right). The mechanism for Eck5 spiking is the same as for Eck6 spiking described above. That is, E_{ff}^{Max} of ENU_5 must occur within the interval $t_k \leq t_{spike} < t_{inter\text{-}inhibitory}$ for Eck5 spiking.

Similar to persistent-Eck6 spiking (Fig. 4.5, bottom left), the Eck5 spiking during B-stimulus (post dual-stimuli) is also continuous (Fig. 4.10b). However, neither Eck5 spiking nor persistent-Eck6 spiking occur in the next trial of B – D&B – B. Figure 4.10c and 4.10d shows that if the duration of dual-stimulus is changed, spiking from Eck5 no longer occurs. Therefore, persistent-Eck6 and Eck5 spiking during B-stimulus following dual-stimuli do not occur frequently as their occurrence is dependent on stimulus instant.

In conclusion, occurrences of persistent-Eck6 and/or Eck5 spiking during B-stimulus following dual-stimuli will not take place if the network does not receive continuous tonic arousal (B-stimulus). Since tonic arousal for either G-N or E-N represents 'alertness' or attention-drive, the likelihood of continuous B-stimulus as a feasible scenario biologically and psychologically requires future investigation. This study establishes that timing and duration of B-stimulus input in this spiking–mode neural network produces emergent network properties that are not captured by the Grossberg-level models. The origin and characterization of the source of B and D inputs is beyond the scope of this thesis.

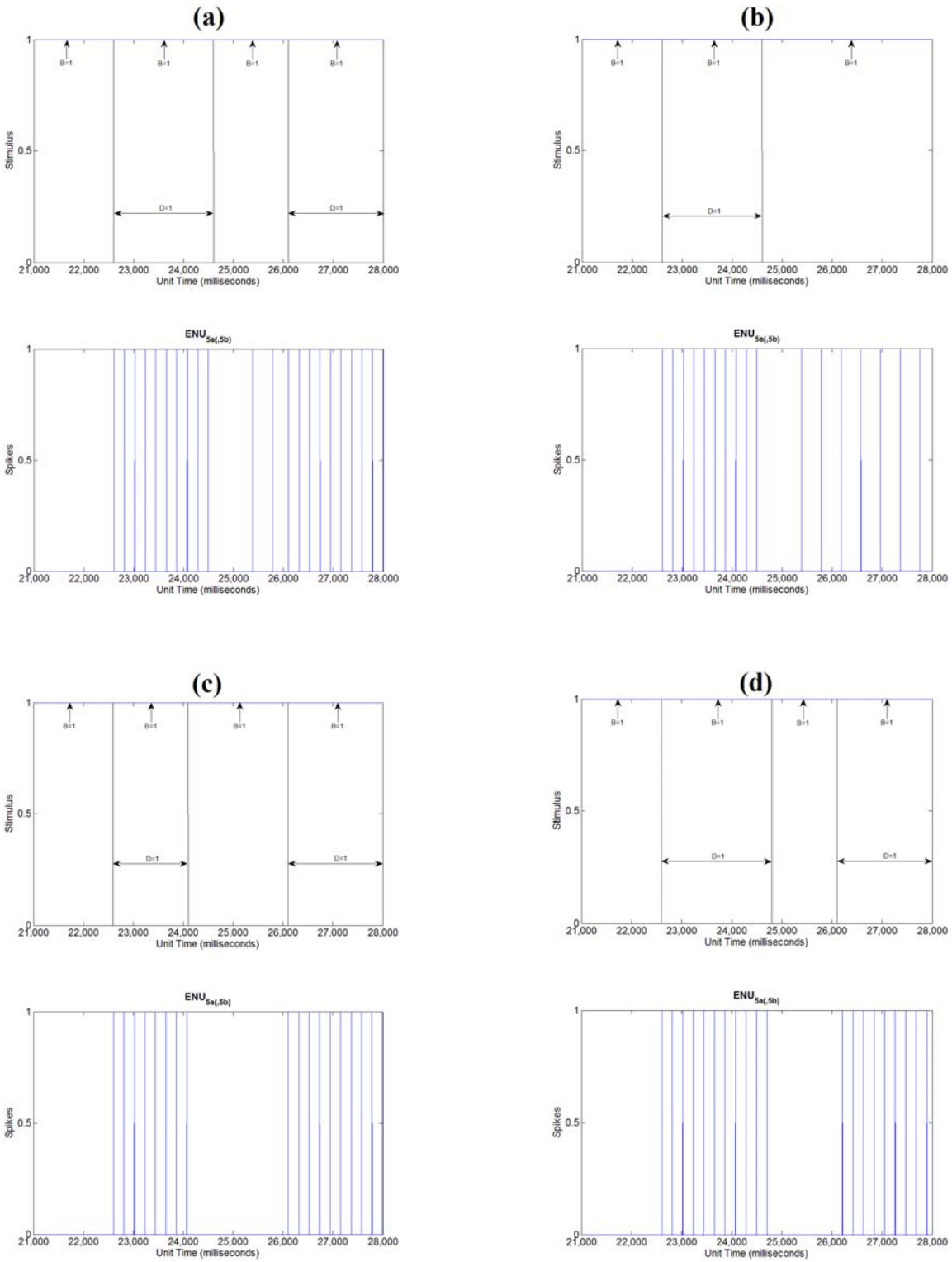

Figure 4.10. a) Simulation seen in Figure 4.2 (bottom right). b) Simulation as in a) but with extended B-stimulus (2nd B of B – D&B – B trial). Notice that Eck5 spiking occurs during B-stimulus following dual-stimuli. c) and d) Simulation with shorter (c) or longer (d) duration of dual-stimuli leads to no Eck5 spiking seen in a) and b).