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A Mathematical Model of Intercranial Electrical Activity and EEG Signal Processing

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Abstract

A dynamic model characterizing the brain signals of an epileptic is simulated numerically. The model is a non-linear, state-space representation involving five variables that control various types of seizures. The electrical activity shows intermittent rapid discharges, spikes, waves and steady time course with possible alternations between normal and ictal (seizure) phases.

The first part of the thesis begins with a model of neural activity during normal and abnormal periods. The primary contribution of the thesis involves numerical solution of the pertinent differential equations using the Runge-Kutta fourth order algorithm. Following this, extensive simulation trials are made to understand the role of the parameters in triggering seizures, what impulsive inputs contribute to the abnormalities, and how they help in restoring the activity to a calm steady state. Several simulated waveforms are included showing the erratic behavior of the brain going through these episodes. Phase-plane plots reveal regions of high intensity seizures distinct from normal resting brain activity. The code was developed and the plots are shown using Matlab software. Of particular significance is the choice of initial conditions. The overall signal behavior can only be explained using ideas from deterministic chaos and bifurcation theory. A clear understanding of the model helps in the development of bio-feedback to control the symptoms. This would be a good topic for future research.

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Acknowledgements

I would like to begin by thanking my wife Kim, and our sons Vincent and Jackson, for their support and confidence. Without their blessing, this thesis would have not been possible. Next, I would like to extend many thanks to Dr. Sudarshan Nelatury. As both my thesis and honors advisor, Dr. Nelatury was with me every step of the way offering guidance and wisdom. There were times in which I was not sure how to proceed and Dr. Nelatury was able to get me back on track with a combination of encouragement, advice and blunt honesty. I am truly appreciative of all these things. The world needs more professors like Dr. Nelatury. Additionally, I would like to thank Dr. Thomas Hemminger for the thoughtful advice I received with respect to my thesis. Dr. Hemminger went far above and beyond my understanding as to the scope of a faculty reader. His interest in his students' success is apparent and much appreciated. Furthermore, I would like to thank Ruth Pflueger for providing much additional guidance to the English aspect of my thesis. Ruth had no obligation to review my thesis but did so out of the kindness of her heart. Lastly, I would like to thank Sharon Hemminger. Sharon first offered me membership to the Behrend honors program. Upon my completion, she encouraged me to apply to the Schreyer Honors College. Sharon helped immensely with my application, and transition, to the Schreyer Honors College. Additionally, she was always there whenever I needed assistance and went out of her way to help me succeed. Without Sharon I would never have been a member of the college in the first place. I had an exceptional group of people who stood behind me and helped me to complete this thesis. Thanks to you all.

Chapter 1

Introduction

Epilepsy is the fourth most common neurological disorder affecting more than 65 million people worldwide. With epilepsy being so common, it is somewhat surprising to learn that little is actually known about this condition. This thesis provides a mathematical model to simulate the most common form of epileptic seizure and presents interesting results that help with in understanding the seriousness of the disorder. In this chapter we begin by providing a general background into epilepsy. This will be followed by a section that provides information about current research including sections on prediction methods and treatment options.

1.1 Background

The director of research at the Institut de Neuroscience des Systme, Christophe Bernard, published an article entitled "Understanding and Predicting Epilepsy" in which he comments "despite decades of research, we do not really know how seizures start, propagate, and terminate, and we still do not understand how a normal brain becomes epileptic." [1, 2]

Epilepsy is neurological disorder in which the sufferer experiences reoccurring bouts of seizures. This activity can be described as sudden abnormal neuronal activity. It can be thought of as an electrical storm in the brain. A precise definition of a seizure becomes somewhat difficult due to the existence of numerous types of events, all of which affect the sufferer in different ways.

The two most common types of seizures are tonic-clonic and absent. A tonic-clonic seizure is the type that people typically think of. It is a convulsive condition that causes the sufferer to shake violently. This type of seizure was until recently referred to as grand mal. An absent seizure is another common type of event. A witness to an absent seizure may describe the sufferer as appearing to be in a trance or a daydream. Someone who is having an absent seizure may freeze in the middle

of an activity for a period of 10 - 20 seconds [3]. Afterward, the sufferer is typically unaware of the episode, or the gap in time. While tonic-clonic and absent seizures are the most common, they are not the only types. There are many more reactions a seizure can elicit on the patient. Additionally, some events affect consciousness and awareness while others do not. A simple seizure is an event in which the person's consciousness and awareness are not affected; whereas a complex seizure affects one or both.

In addition to the differing physical manifestations, there can be a great deal of variability with respect to region(s) of the brain affected in epileptic patients. A partial, or focal, seizure is one in which abnormal neural activity is limited to a single hemisphere of the brain whereas a general seizure affects both hemispheres. Sometimes this will begin in one region of the brain and spread to another. It is also possible to have partial onset then propagate to the other hemisphere of the brain. This is referred to as a secondary generalized seizure.

Epileptic symptoms are classified by three considerations; where it starts, if consciousness and awareness are affected, and whether it involves movement [2]. For example a complex partial tonic-clonic seizure would begin in a single region of the brain and affect the consciousness and awareness of the sufferer. Further, this classification indicates that the person would experience convulsions. A complex partial seizure is the most common type of event experienced by adult epileptics [4] and will be the focus of this work.

1.2 Current Research

Several areas of research have emerged in an effort to develop methods to combat the disease. Considerable effort has been dedicated to understanding the dynamics that drive seizures, methods to predict and classify neural activity, and treatment options.

1.2.1 Prediction and classification

Methods to predict and identify this condition first rely on obtaining physiological data. Most commonly, an electroencephalogram (EEG) is used to monitor activity in the brain. Traditionally, data is collected and interpreted by a neurologist who determines if the data resembles a seizure. However, traditional methods lend themselves to drawbacks such as human error. A solution to this problem is to limit, or remove, the need for human interpretation. To achieve this; the data must be analyzed and classified using computers.

A solution to overcome human error in interpreting EEG data was proposed by Sutrisino Ibrahim et al. The proposed method combines the observations of a neurologist with computer aided diagnosis (CAD). The EEG signal is decompressed using a discrete wavelet transform (DWT). Shannon entropy is then used to measure the distribution of the data [5]. The information obtained from this method is then compared to prerecorded baselines that represent normal brain activity and neural activity. Variances with regard to both normal and epileptic brain activity between patients require individual baselines to be obtained. An algorithm, K-nearest neighbors, is used to

determine which baseline the signal more closely matches. The results are used as a suggestion to the neurologist to guide his/her interpretation. The neurologist is then able to provide feedback regarding the accuracy of the suggestion made by the CAD system. This feedback is then used to update the original information, thus improving future CAD recommendations. A benefit to this method is that it can either give the neurologist confidence in diagnosis, or it can encourage a closer look at the data. A drawback is that a neurologist is still required at early stages of the study and accurate individual baselines must be obtained before utilizing this method.

Gautam et al. in [6] propose a method to distinguish between focal and general onset seizure using signal processing methods. Visual imaging methods such as computerized tomography (CT) or positron emission tomography (PET) scans are typically employed to determine the regions of the brain affected by an epileptic event but at a cost of a dose of radiation.

The approach produces similar results without the need for radioactive imaging methods such as CT or PET scans. It relies on empirical mode decomposition to extract intrinsic mode functions (IMF) from EEG data. Intrinsic mode functions are a finite set of amplitude and frequency modulated components [6]. Deriving an IMF is an iterative process in which the next IMF is determined from the current result. The iterations continue until all solutions have been found and no further information can be extracted. Once all IMFs have been determined, a comparison of peak values can be made. This comparison can help identify the type of seizure. Results of this method are shown in Figure 1.1.

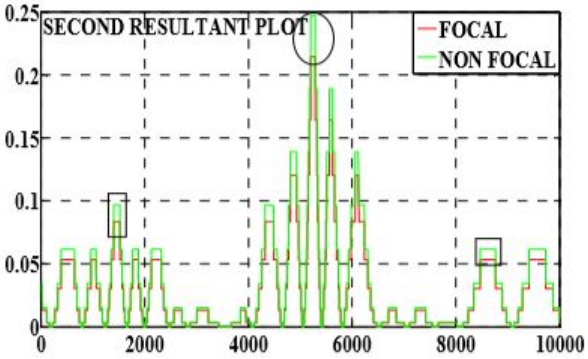

Figure 1.1: Classification of focal vs. non-focal seizure using EMD [6]

The EEG is not the only signal that can be analyzed to predict seizures. An electrocardiogram (ECG) can also be used to identify an event based on the rhythm of the heart. This prediction is based on variability in heart rate. The benefit to this method of detection is that the subject can place a patch on the chest to monitor heart rate. A device connected to the patch can then be used to identify potential epileptic activity.

The heart produces a rhythm which is characterized by five peaks identified as P, Q, R, S, T with the R peak being the largest. Figure 1.2 shows a single period of a heart rhythm with peaks identified. Heart rhythm is predictable and specific variances in the normal heart rhythm are indicative of seizure onset. Heart rate variability is typically measured by considering the change in

the interval between R-peaks. Jeppesen et al. propose an algorithm for the detection of R-peak variability in [7]. The proposed algorithm adaptively adjusts the threshold used to distinguish R-peaks from other peaks while monitoring R-peak variability.

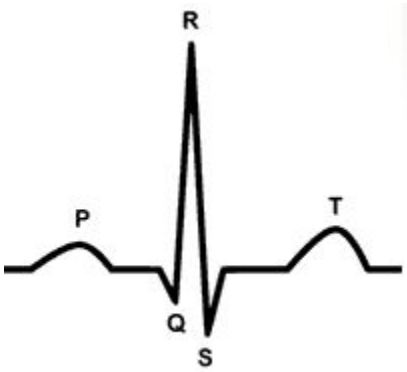

Figure 1.2: Heart wave with labeled peaks

An issue with using heart rate variability to detect seizures is the occurrence of false positives. False positives can be attributed to activities that affect heart rate variability in a similar way as a seizure-like event. Activities such as exercise can trigger a false positive. A method to overcome this is proposed by Jeppesen et al. in [8]. The proposed method begins by detecting the R-peaks using an existing algorithm. A Lorenz plot is then constructed by plotting each R-R peak interval against the next R-R peak interval [8]. Elongation along the transverse axis indicates the occurrence of a seizure as shown in Figure 1.3.

It has been noted that in the late pre-seizure and early seizure state there was an increase in the longitudinal component of the Lorenz plot. They then modified the plot to emphasize the longitudinal component thus excluding false positives from the resultant plot. Exercise activity is shown in Figure 1.4. Note the tight grouping of data points indicating exercise activity versus the elongated longitudinal grouping indicative of seizure activity.

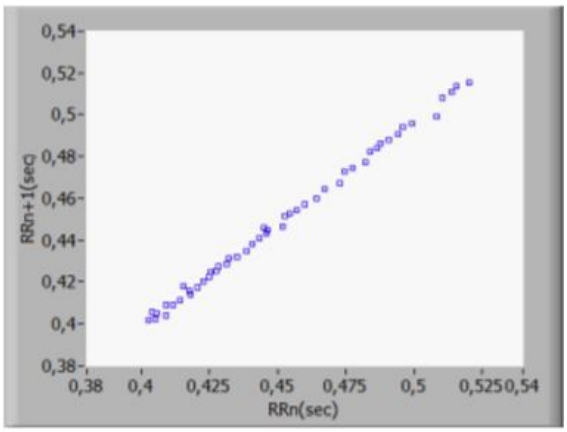

Figure 1.3: Lorenz plot showing seizure activity [8]

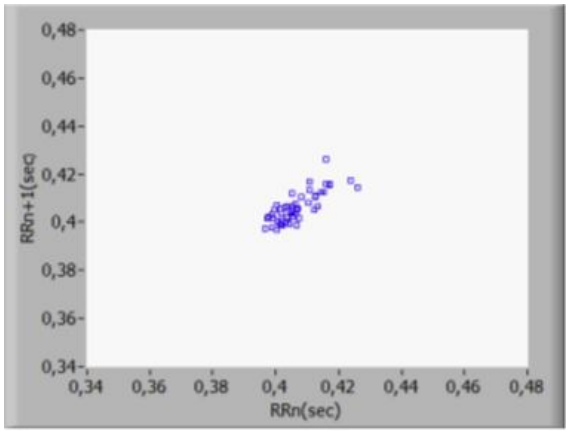

Figure 1.4: Lorenz plot showing exercise activity [8]

There has been much research conducted to forward the state of seizure prediction and classification. The few examples discussed in this section are only a small part of a much bigger picture. One big challenge is the short time in which vast amounts of data is required to be processed.

1.2.2 Treatment

Treatment of epilepsy presents many challenges. The predominant class of seizure caused by epilepsy is the complex partial seizure. Unfortunately, this is also the type of event that is most likely to develop a resistance to medications over time. Neurostimulation may be an alternative when drugs fail or are not an option.

An open loop neurostimulator is a device that is typically implanted in the chest of an epileptic patient. A pair of wires run under the skin from the device to a set of electrodes that are either implanted in the brain or attached to the vagus nerve in the neck. When the patient, or caregiver, becomes aware of an imminent, or active seizure, a magnetic card can be placed over the device externally to activate it. Once it has been activated, an electrical impulse is delivered to the patient. Successful operation of the neurotransmitter results in termination of the seizure.

A drawback to open loop stimulation is that it requires preprogrammed parameters that define the amplitude, frequency, and duration of the pulse train delivered to the patient [9]. The neurotransmitter is not capable of adjusting these parameters to differences in electrical activity in the brain. This inability can result in a pulse train being delivered that is either inadequate or excessive. Another major drawback to open loop neurostimulators is that they require someone to physically activate them.

Closed loop neurostimulation is the latest technology. A sensor is used to monitor the patient for seizure activity. The sensor is typically a thin mesh grid that is placed directly on the surface of the brain. The grid communicates with a processor that is implanted in the chest of an epileptic patient. Communication can either take place along a pair of conductors implanted in the patient's body that connects the grid directly to the processor or by using a wireless protocol. The mesh

grid and processor function together to act as an EEG. The system monitors the electrical activity for seizures. Once a seizure is detected, it determines the size, shape, and duration of the pulse to be delivered to the patient. A signal is sent from the processor to the mesh grid which delivers the pulse. The system then monitors the response and will prescribe another pulse to be delivered if necessary [10].

While great advances have been made with regard to epileptic research, there is still much work to be done. As mentioned earlier, we still know very little about the dynamics of a seizure [1]. Most of the research discussed thus far applies to the care and treatment of epilepsy. A different approach is needed to get to the heart of what fuels this disorder. One such approach includes the construction of a model. This approach is introduced in the next chapter.

Chapter 2

Epileptor

The epileptor is a mathematical model of the electrical activity in a brain prior to, during and after a seizure. It was developed by a group of researchers led by V. Jirsa and C. Bernard and presented in a 2014 paper entitled "On the Nature of Seizure Dynamics." It was developed to help provide a better understanding of the dynamics of this type of brain activity. It consists of a set of five state space equations which are highly dependent on each other and highly non-linear. This chapter will discuss the epileptor equations and the methods used to reproduce them.

2.1 Equations

To develop the epileptor equations, Jirsa et. al. first separated an EEG representative of seizure activity into two components they called ensembles. The first ensemble represents fast discharges. These fast discharges are high frequency oscillations. The second ensemble represents what is referred to as spike and wave events. These spike and wave events consist of high amplitude spikes. Figure 2.1 shows the EEG of a mouse placed in a preservative seizure state. The figure is labeled to illustrate the difference between the spike and wave ensemble and the fast discharge ensemble [11].

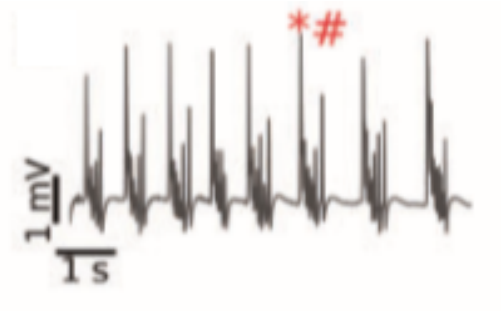

Figure 2.1: EEG from a mouse placed in a persistent seizure. # represents fast discharges, * represents spike and wave event [11]

Once the two ensembles were classified, the goal was to develop a set of state space variables to describe them mathematically. It was noted that the onset, and offset, of seizure activity deviated from normal activities in distinct ways. These deviations are expressed as two bifurcations. Bifurcations are changes that occur in a dynamic system as the result of a change to some parameter. In this case, these bifurcations closely resemble two known patterns which are saddle-node at onset and homoclinic at offset. This particular combination has been well studied and is more commonly referred to as a square wave burster. Using previously derived equations for the square wave burster as a starting point, modifications to relate the two ensembles were made through the addition of another state space variable which essentially serves as a time base for the epileptor. This time base variable is dependent on the other two ensembles. Additionally, the two ensembles are dependent on the time base variable as well. The precise derivation for the epileptor was not provided; however, the equations and all relevant parameters are provided [11].

The state space equations for the epileptor are shown below in equations 2.1 - 2.5. The x_1 and y_1 variables represent the fast oscillation ensemble. The x_2 and y_2 variables represent the spike and wave ensemble and the z variable represents the time scale [11]. The epileptor is plotted on a three-dimensional axis defined by $-x_1$, x_2, z. The field potential, which resembles EEG activity, is constructed by plotting $-x_1 + x_2$ as a function of time.

$$\dot{x}_1 = y_1 - f_1(x_1, x_2) - z - I_{rest1} \tag{2.1}$$
$$\dot{y}_1 = y_0 - 5x_1^2 - y_1 \tag{2.2}$$
$$\dot{z} = \frac{1}{\tau_0}(4(x_1 - x_0) - z) \tag{2.3}$$
$$\dot{x}_2 = -y_2 + x_2 + x_2^3 + I_{rest2} + .002g(x_1) - 0.3(z - 3.5) \tag{2.4}$$
$$\dot{y}_2 = \frac{1}{\tau_2}(-y_2 + f_2(x_1, x_2)) \tag{2.5}$$

where;

$$g(x_1) = \int_{t_0}^{t} e^{-\gamma(t-\tau)} x_1(\tau) d\tau \tag{2.6}$$

$$f_1(x_1, x_2) = \begin{cases} x_1^3 - 3x_1^2, & x_1 < 0 \\ x_1(x_2 - 0.6(z-4)^2), & x_1 \geq 0 \end{cases} \tag{2.7}$$

$$f_2(x_1, x_2) = \begin{cases} 0, & x_2 < -0.25 \\ 6(x_2 + 0.25), & x_1 \geq -0.25 \end{cases} \tag{2.8}$$

Initial conditions are crucial to obtaining a valid result to a differential equation. Initial conditions are known values of a solution to a differential equation at a given time. The epileptor defines initial conditions as follows: $x_1 = 0,\ y_1 = -5,\ z = 3,\ x_2 = 0,\ y_2 = 0.$

2.2 Reproduction

The epileptor was reproduced using the fourth order Runge-Kutta method to approximate the solution to the differential equations. Runge-Kutta is an iterative method that is dependent on previous values and initial conditions. The equations that are used for Runge-Kutta are shown in equations 2.9 - 2.15.

$$y_{n+1} = \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4) \tag{2.9}$$

$$\tag{2.10}$$

where;

$$k_1 = h\ f(t_n, y_n) \tag{2.11}$$

$$k_2 = h\ f\left(t_n + \frac{h}{2}, y_n + \frac{k_1}{2}\right) \tag{2.12}$$

$$k_3 = h\ f\left(t_n + \frac{h}{2}, y_n + \frac{k_2}{2}\right) \tag{2.13}$$

$$k_4 = h\ f(t_n + h, y_n + k_3) \tag{2.14}$$

$$t_{n+1} = t_n + h \tag{2.15}$$

Matlab was then used to carry out the computation of the Runge-Kutta method. Because the equations of the epileptor are highly dependent on each other and contain piecewise functions within, the standard preprogrammed Matlab differential equation solvers were not used. Instead, a new script was written to carry out the computation. Figure 2.2 shows a plot of the original epileptor obtained from [11]. A high degree of similarity is noted when compared with the reproduced epileptor shown in Figure 2.3. The discrepancies are attributed to two factors. The first is due to

differing methods used to approximate the solutions to the equations. The second is due to the addition of Gaussian white noise in the original which was not included in the reproduction.

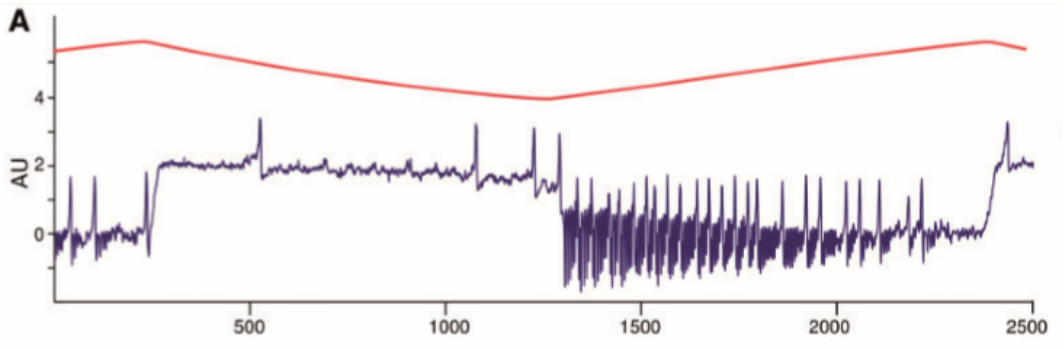

Figure 2.2: Original epileptor [11]

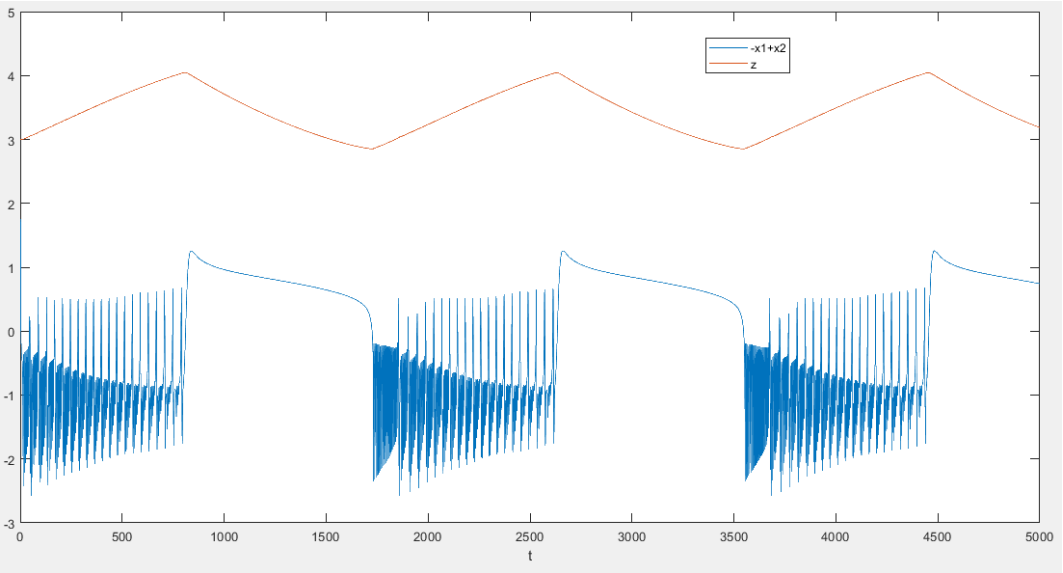

Figure 2.3: Reproduction of epileptor

Chapter 3

Working With the Epileptor

This chapter describes modifications that were made to the defined initial conditions in an effort to gain a better understanding of the role individual state variables play in the epileptor. Once a firm understanding has been developed regarding the role of individual state variables, the knowledge gained is then used to alter the algorithm. First, an electrical pulse is delivered with the intent of driving the epileptor to early seizure onset. Next, a second electrical pulse is employed in an attempt to terminate the seizure.

3.1 Modification to Initial Conditions

In order to better understand the role the initial conditions play in this paradigm, modifications are made and the resultant waveform is observed. Figure 3.1 shows the first two seizure periods. This plot will be used to compare future plots of modified initial conditions.

3.1.1 Modification to z

The time scale of is based on the z variable, which is defined in equation 2.3. It is the z variable which is responsible for triggering both the onset and offset of a seizure. The onset of a seizure occurs at the point where z transitions from having a negative slope to a positive slope. This transition occurs when z reaches a minimum value of approximately 2.85. A seizure offset occurs at the point where z transitions from a positive to a negative slope. This transition occurs when z has reached a maximum value of approximately 4.05.

Modifications to the initial condition of the z variable were made to gain an understanding of the effect on the overall output of the epileptor. First the initial condition was increased from

$z_0 = 3$ to $z_0 = 4$. The resultant plot shown in Figure 3.2 shows that an increase in the initial condition causes a shift in seizure onset. The overall shape of the waveform is unaffected, indicating that the only significant change is a shift in time.

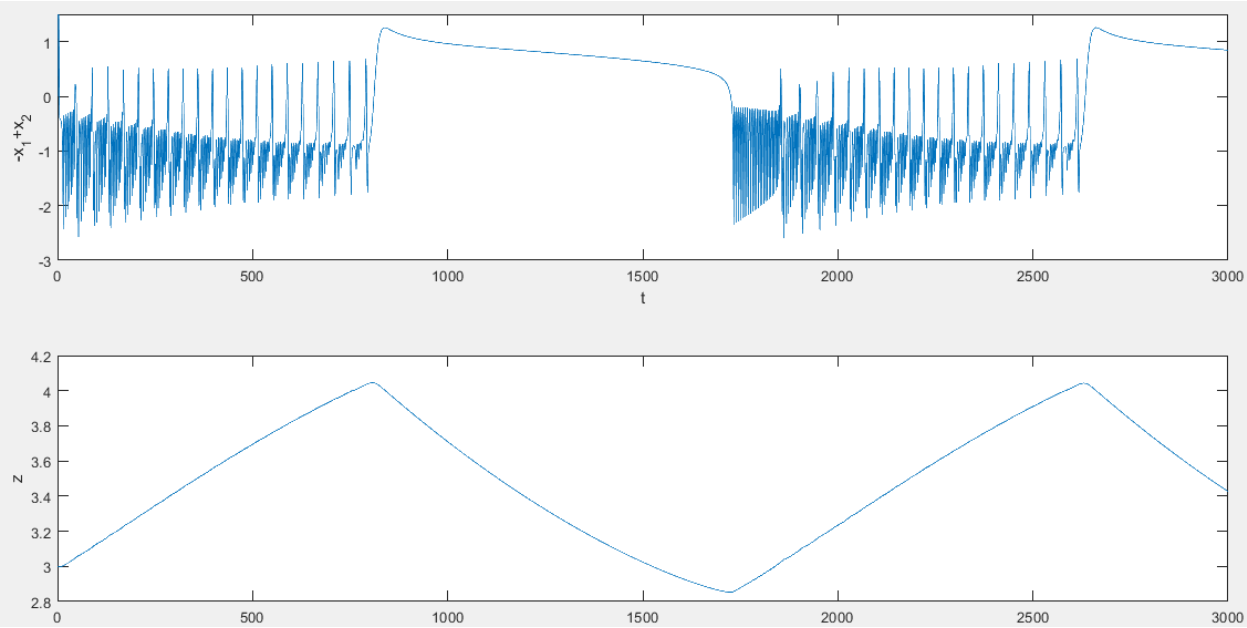

Figure 3.1: First two seizures derived from unmodified epileptor. The upper plot depicts a seizure while the lower plot shows the time scale z

Increasing the initial condition in z causes the slope of the plot of z to be inverted. It can be seen that the plot of z in Figure 3.2 is approximately equal to the inverse of the plot of z in the original output shown in Figure 3.1. Additionally, once the first event has occurred, the time interval between successive seizure onset is constant and equal to the interval in the original. Therefore, the initial value can be increased indefinitely without causing the epileptor to breakdown. The only consequence to increasing the initial condition of z is a delay in the onset of the initial seizure.

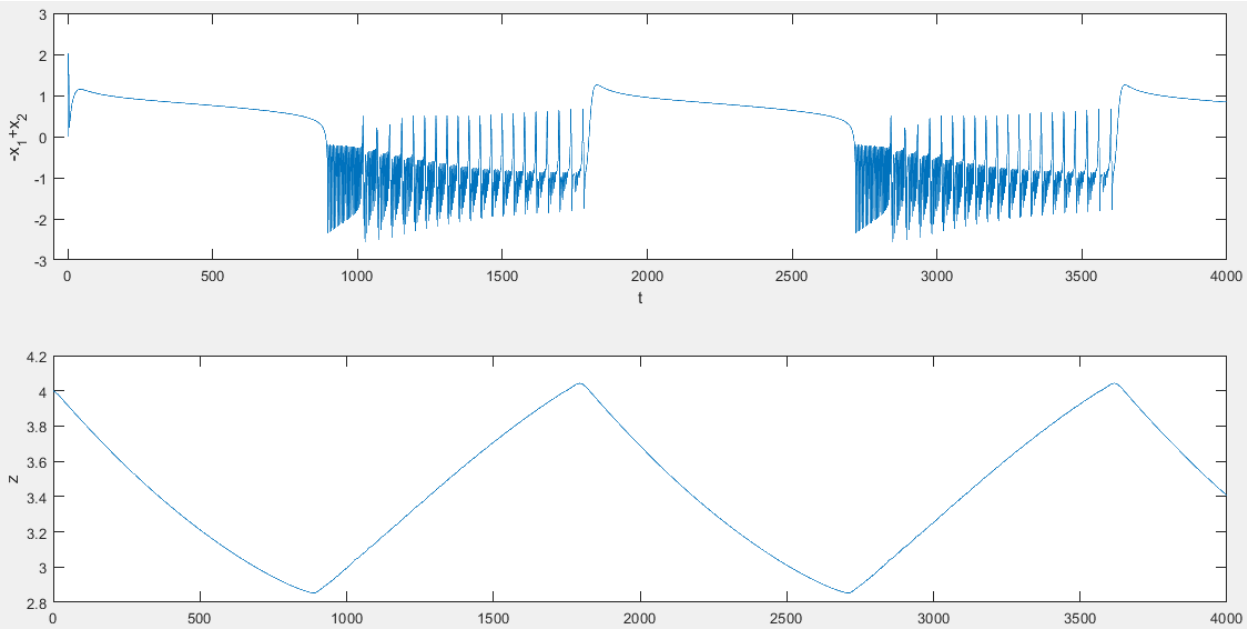

Figure 3.2: Effect of increasing the z initial condition on the epileptor

Next, the initial condition of z is decreased from $z_0 = 3$ to $z_0 = 2$. The resultant plot shown in Figure 3.3. It is shown that the initial seizure occurs at the same time the initial event occurred in the unmodified model; however, there is an increase in duration. This increase in duration is due to the fact that the z variable begins below the normal minimum value for z of roughly 2.85. It is also noted that decreasing below this minimum causes the slope of z to decrease. This decrease in slope is temporary and eventually returns to the original slope. During this time, the signal consists of only a high frequency component and no longer contains any abrupt spikes.

The initial condition was further decreased to explore the impact that the change in initial slope had on neural activity. Decreasing the initial condition of z from $z_0 = 2$ to $z_0 = 1.5$ causes the epileptor to become highly unstable and eventually diverge. The resultant plot is shown in Figure 3.4. Any further decrease causes the epileptor to no longer exist.

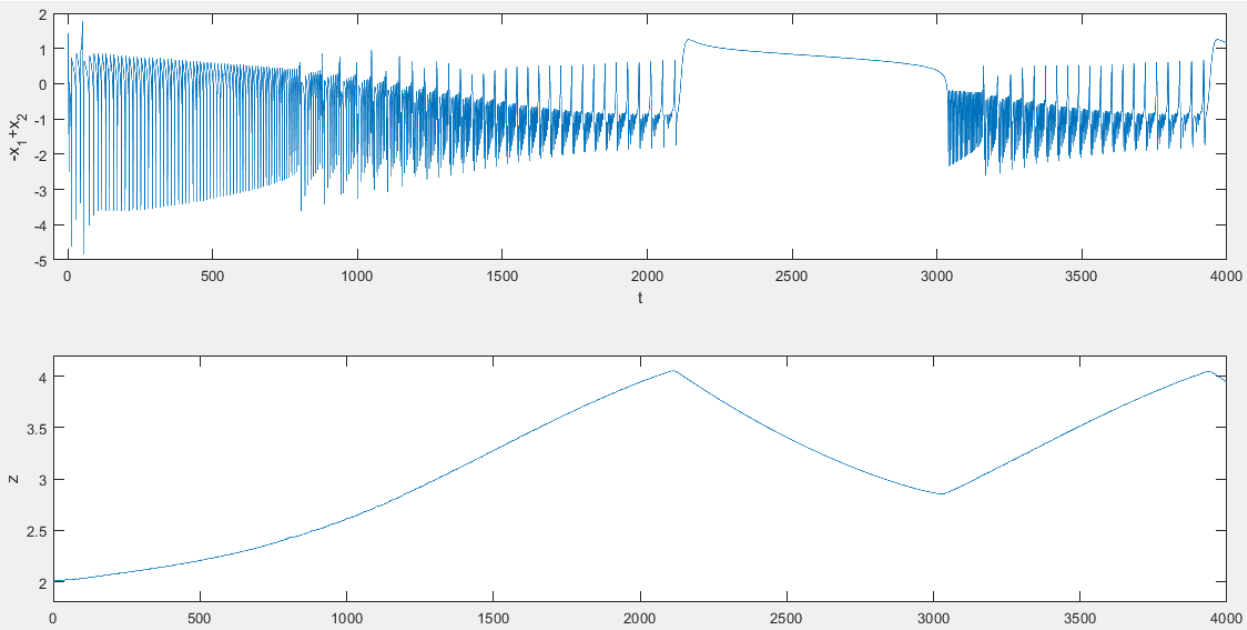

Figure 3.3: Effect of decreasing the z initial condition on the epileptor

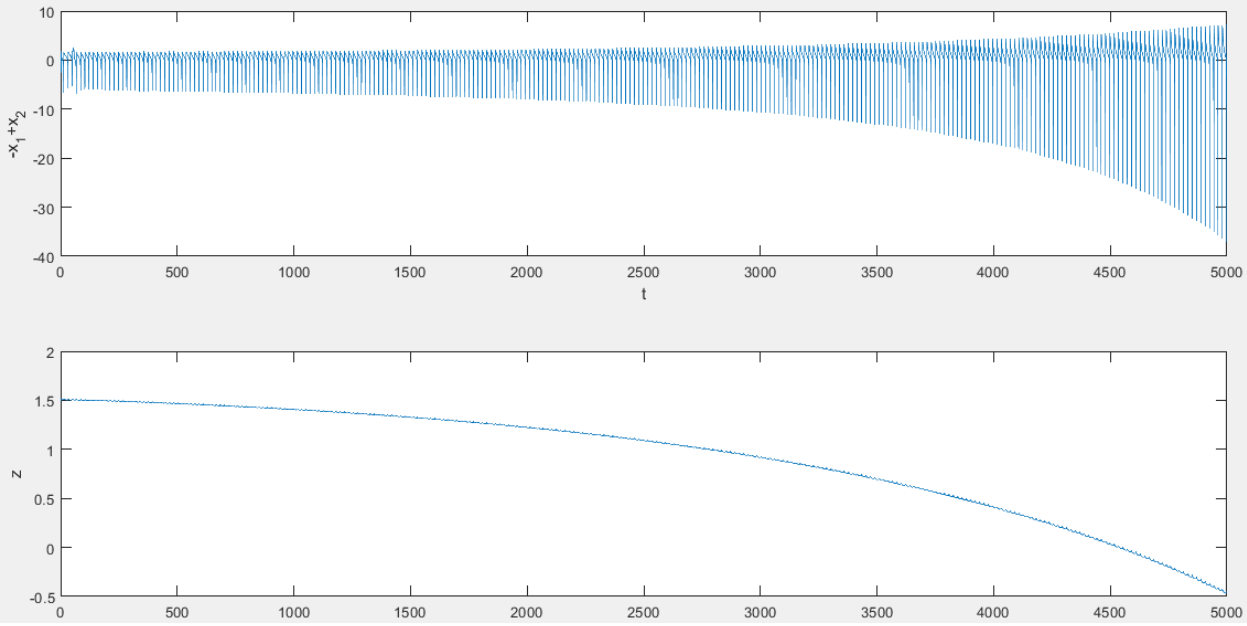

Figure 3.4: Effect of decreasing the z initial condition on the epileptor to the point of breakdown

3.1.2 Modification to x_1 and y_1

According to [11], state variables x_1 and y_1 are responsible for the high frequency component of the model. Adjustments to the initial conditions for the variables x_1 and y_1 are made to gain a better understanding of how these variables affect it.

To begin, the initial condition of the variable x_1 is adjusted from $x_{1\_0} = 0$ to $x_{1\_0} = 5$. The resultant plot shown in Figure 3.5 reveals that the adjustment causes a shift in the time to initial seizure onset. Comparing to the original plot shown in Figure 3.1, we can see that instead of z beginning with a positive slope, it now begins with a negative slope. This change in slope is what is responsible for the delay in initial epileptic onset. This result was unexpected; however, it serves to show how highly dependent the state variables are on one another.

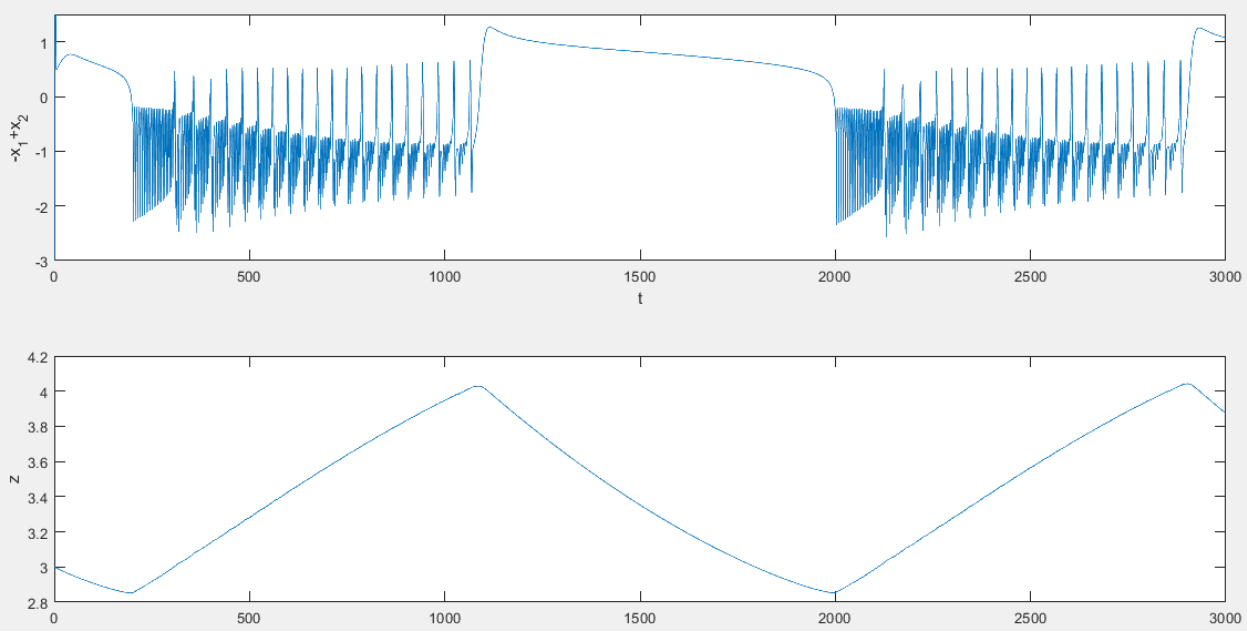

Figure 3.5: Effect of increasing the x_1 initial condition on the epileptor

A negative adjustment to the x_1 initial condition is then made by changing $x_{1\_0} = 0$ to $x_{1\_0} = -5$. The resultant plot shown in Figure 3.6 bears striking resemblance to the plot obtained by increasing the initial condition of x_1. Surprisingly, decreasing the initial conditions to x_1 had the same effect as increasing the initial conditions of x_2. Even more surprisingly, adjusting the initial conditions to x_1 in either direction had no impact on the high frequency component of the seizure for which this variable is responsible. Instead, the major change was due to the time scale variable z.

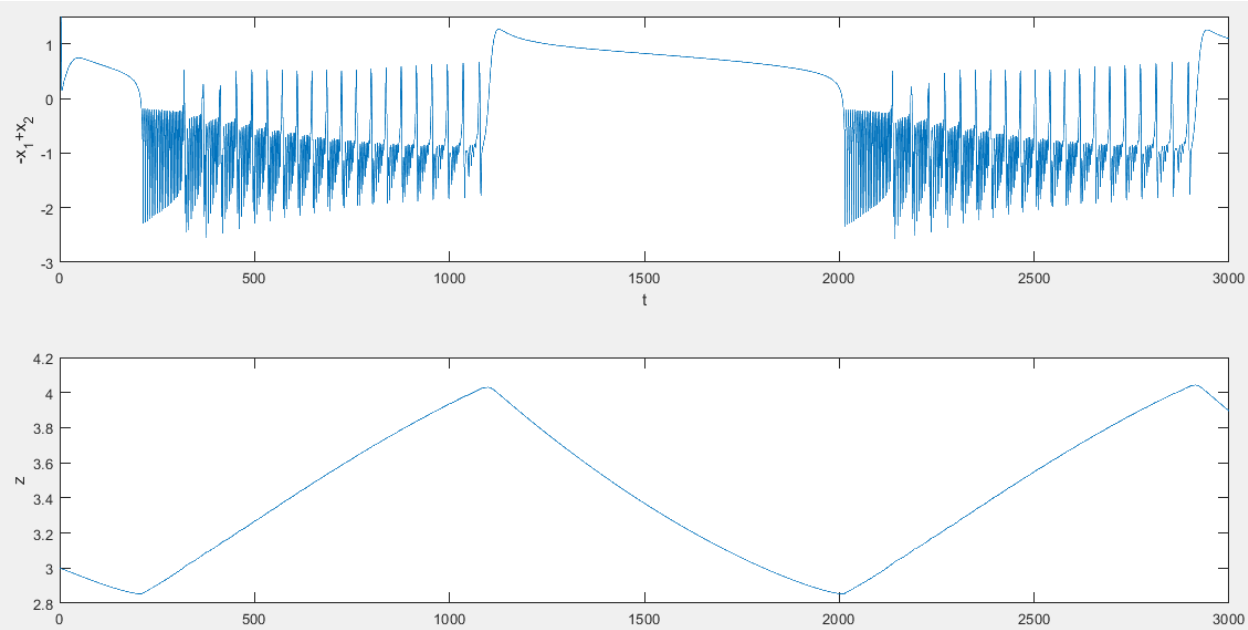

Figure 3.6: Effect of decreasing the x_1 initial condition on the epileptor

The other variable responsible for the high frequency component of the epileptor is the y_1 variable. The initial conditions of this variable were then adjusted and observed. First, the initial conditions were adjusted by the same magnitude used to adjust x_1 and were adjusted from $y_{1\_0} = -5$ to $y_{1\_0} = 0$. The resultant plot shown in Figure 3.7 shows no identifiable difference to the original plot in Figure 3.1. Increasing the magnitude of the adjustment to $y_{1\_0} = 10$ caused the plot to become virtually identical to the plot obtained from increasing the x_1 variable. The resultant plot shown in Figure 3.8 shows that, similar to Figure 3.5, the onset of the initial seizure is delayed. Further, it is noted that the initial slope of z has gone from positive to negative.

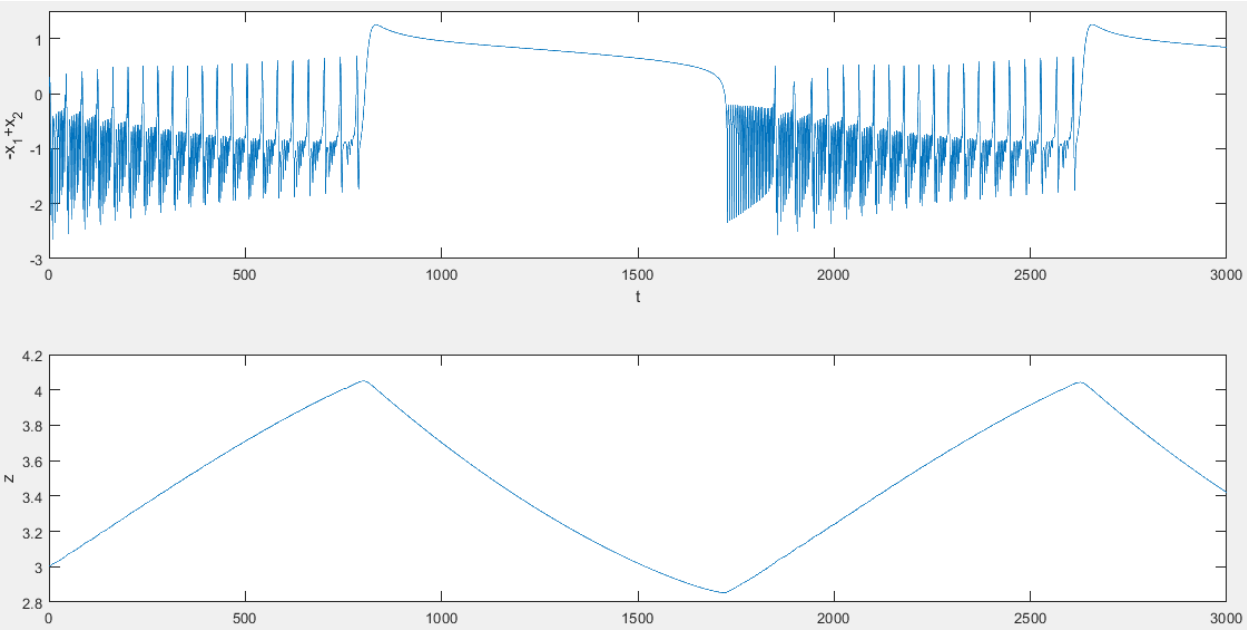

Figure 3.7: Effect of increasing the y_1 initial condition on the epileptor

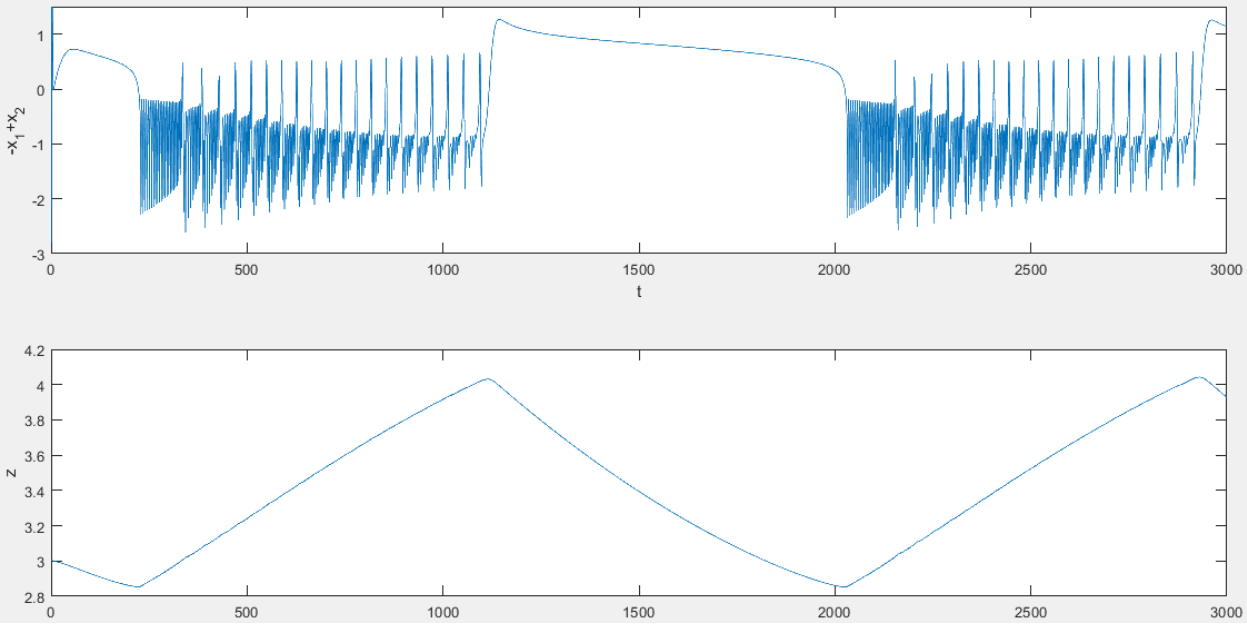

Figure 3.8: Effect of continuing to increase the y_1 initial condition on the epileptor

The y_1 initial condition is then decreased from $y_{1\_0} = -5$ to $y_{1\_0} = -10$. The resultant plot shown in Figure 3.9 indicates that a decrease to y_1 has the same effect as increasing y_1. It is noted that the initial slope of z is negative and that the onset of the initial seizure has been delayed.

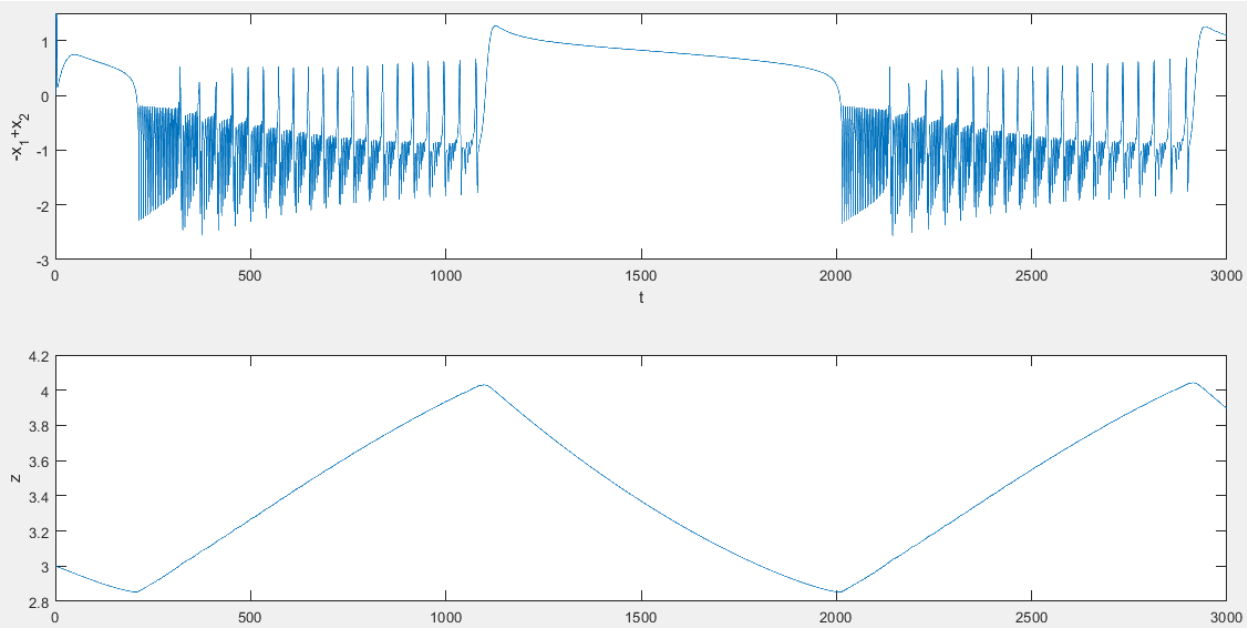

Figure 3.9: Effect of decreasing the y_1 initial condition on the epileptor

The effect of modifying the initial condition of x_1 is the same as modifying the initial condition of y_1. While both of these variables contribute to the high frequency components of the epileptor, adjustment to the initial conditions has no effect on this portion of the signal. Rather, adjustment in either direction to either variable causes a change to the time scale variable z. This change causes the onset of the initial seizure to be delayed. There is no amount of adjustment in either the positive, or negative, direction that causes the epileptor to become unstable. Further, the time interval by which the seizure is delayed remains constant (once the initial shift has occurred) regardless of the magnitude of change to the initial conditions.

3.1.3 Modification to x_2 and y_2

The x_2 and y_2 variables are responsible for producing the spike features that have been observed. To determine the role the individual x_2 and y_2 variables play, an adjustment is made to the initial conditions of one while holding all others constant. Fist, an adjustment is made to the x_2 variable by changing $x_{2\_0} = 0$ to $x_{2\_0} = 15.48$. The resultant plot shown in Figure 3.10 shows that this adjustment has no effect on the epileptor. However, increasing x_{2_0} by 0.01 to $x_{2\_0} = 15.49$ causes severe instability. This instability causes breakdown which is shown in Figure 3.11.

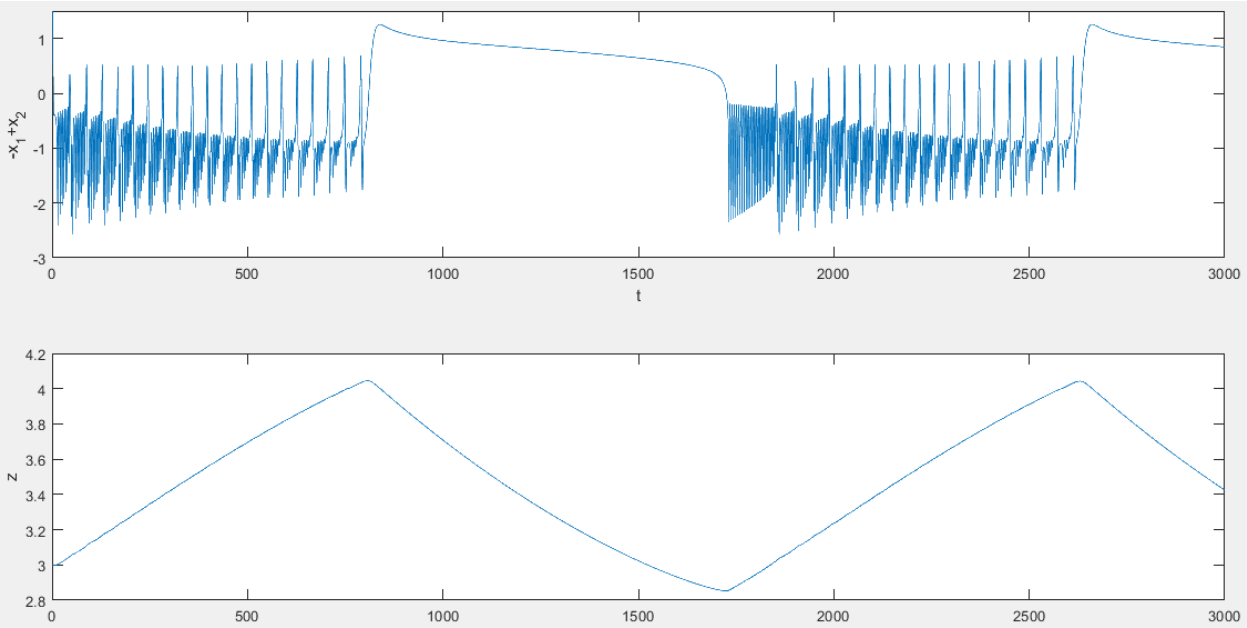

Figure 3.10: Effect of increasing the x_2 initial condition on the epileptor

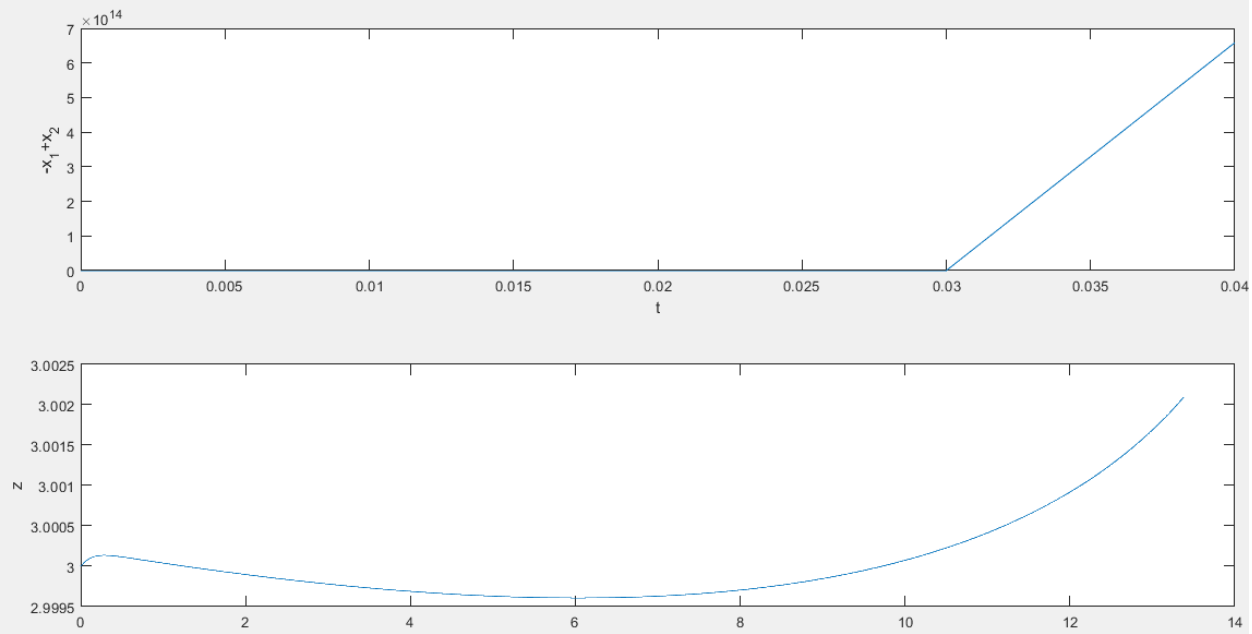

Figure 3.11: Effect of increasing the x_2 initial condition on the epileptor to the point of breakdown

Next, the effect of a decrease to the initial conditions of the x_2 variable is explored. A negative adjustment to this variable is shown to have a similar effect as was observed by the positive adjustment. The variable x_2 is reduced from $x_{2\_0} = 0$ to $x_{2\_0} = -15.49$. It is shown in Figure 3.12 that the output of the epileptor has not changed from the original version. However reducing the

initial condition by 0.01 to $x_{2\_0} = -15.50$ causes the epileptor to become unstable and breakdown. Figure 3.13 shows the breakdown caused by reduction to the x_2 initial condition.

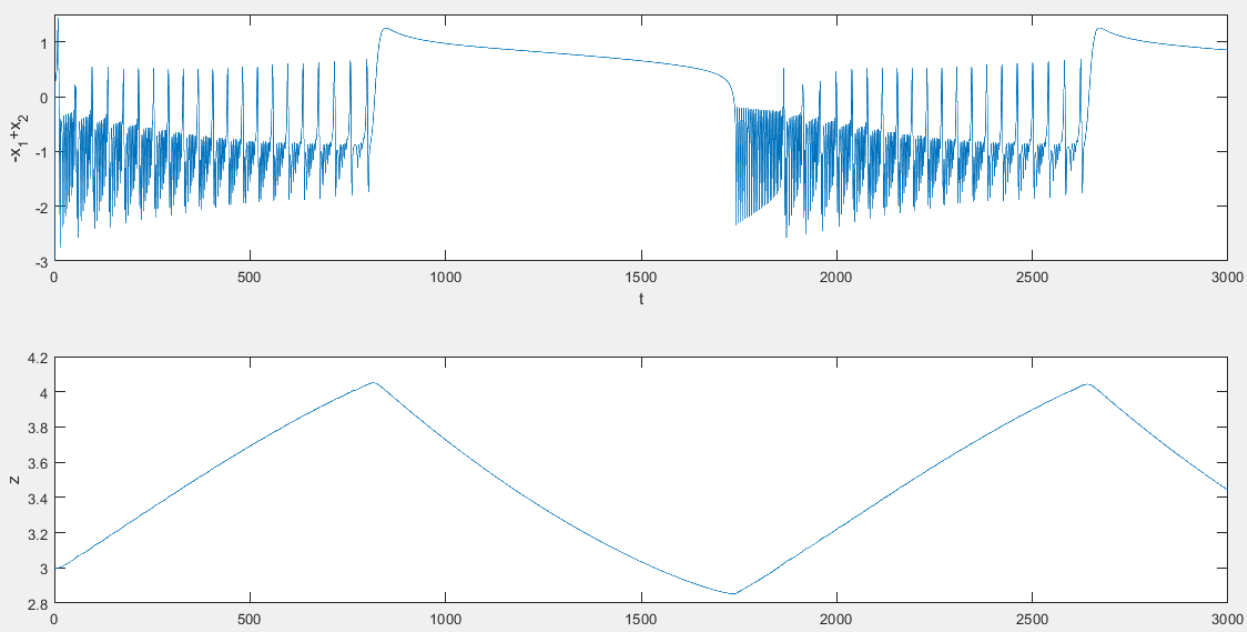

Figure 3.12: Effect of decreasing the x_2 initial condition on the epileptor

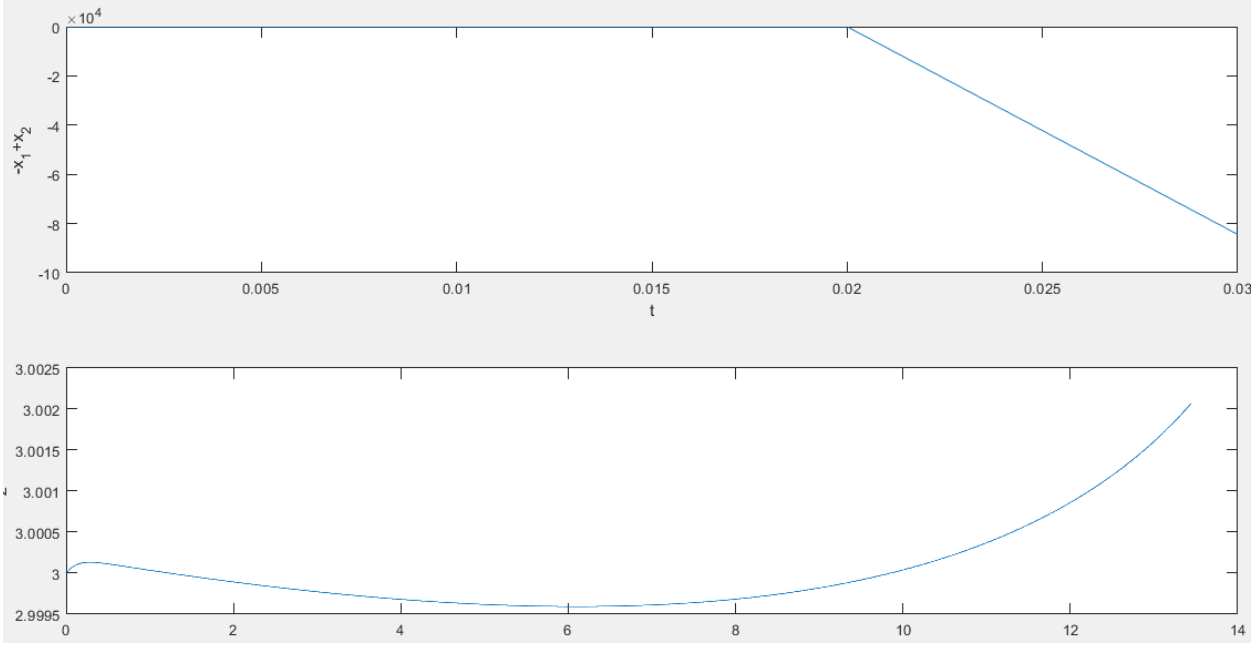

Figure 3.13: Effect of decreasing the x_2 initial condition on the epileptor to the point of breakdown

The modification to the x_2 initial condition was found to have no effect on the output of the system until the adjustment reached a point where a small increase in magnitude it to breakdown.

Adjustment was then made to the y_2 variable to determine if it behaved in a similar fashion. Surprisingly, adjustment to this variable caused a similar effect to adjusting the x_1 and y_1 variables. Figure 3.14 shows the effect of adjusting $y_{2\_0} = 0$ to $y_{2\_0} = 20$. This plot shows remarkable similarity to the plots that were created by adjusting the x_1 and y_1 initial conditions. It was found that the epileptor becomes unstable when the initial conditions are raised to a high value. Figure 3.15 shows the point where breakdown occurs at $y_{2\_0} = 1823$.

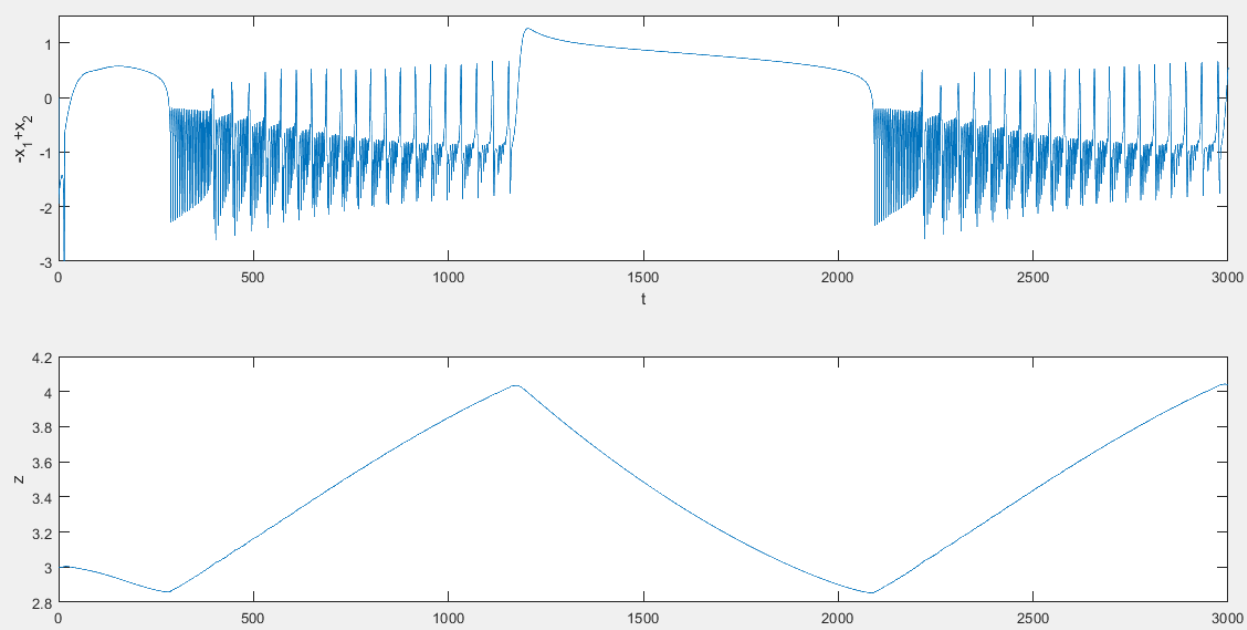

Figure 3.14: Effect of increasing the y_2 initial condition on the epileptor

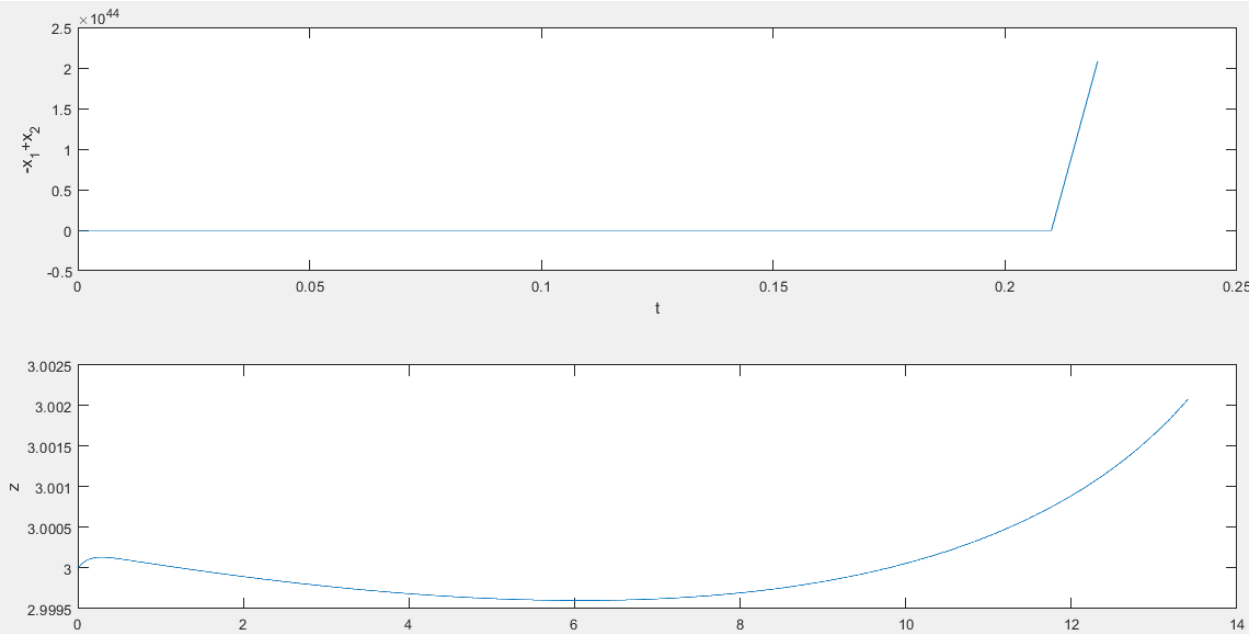

Figure 3.15: Effect of increasing the y_2 initial condition on the epileptor to the point of breakdown

A negative adjustment was then made to the initial conditions of the y_2 variable. Figure 3.16 shows that the epileptor output is unaffected by decreasing $y_{2\_0} = 0$ to $y_{2\_0} = -20$. In fact, the output is completely unaffected until the initial conditions reaches a point where a small decrease results in total breakdown. This point is shown in figure 3.17 where $y_{2\_0} = -1822$.

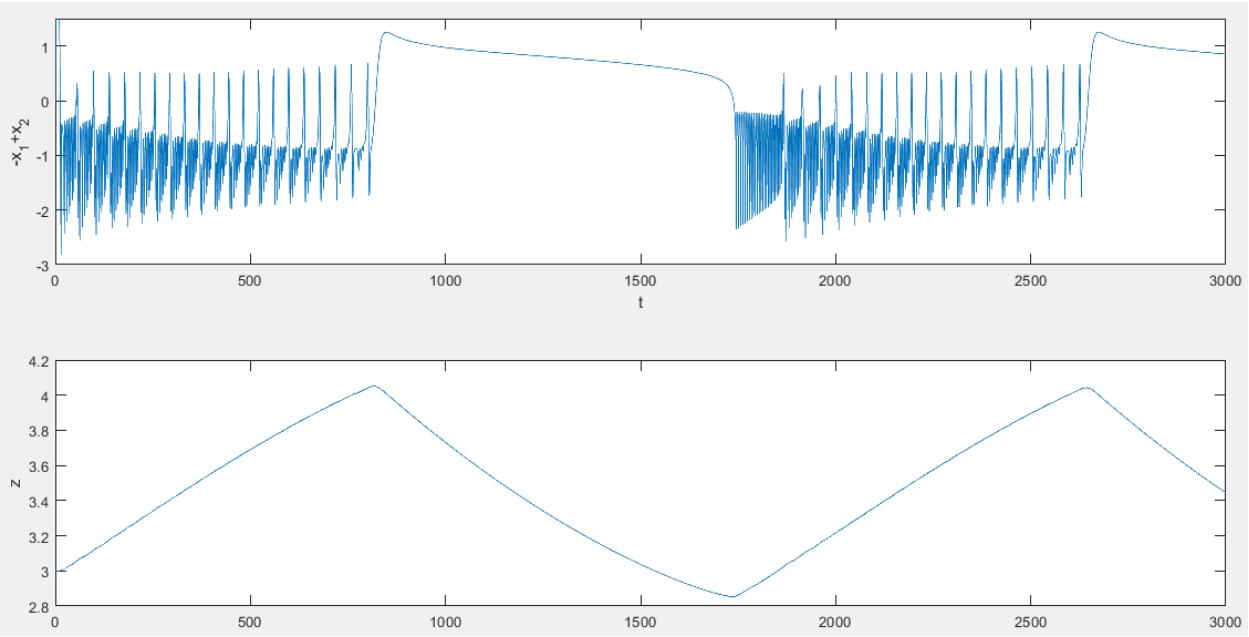

Figure 3.16: Effect of decreasing the y_2 initial condition on the epileptor

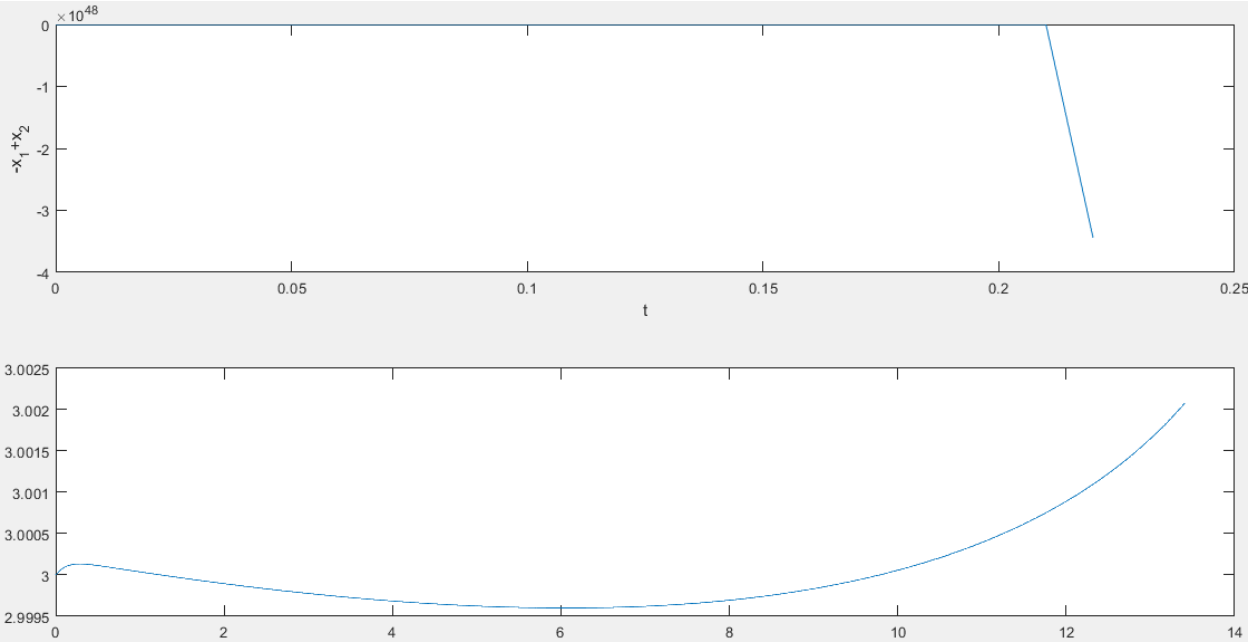

Figure 3.17: Effect of decreasing the y_2 initial condition on the epileptor to the point of breakdown

Overall, adjustments to either the x_2 or y_2 initial conditions have little effect until a further small increase of respective magnitude causes the epileptor to breakdown. Aside from this, the only significant deviation from the original plot (Figure 3.1) occurs when y_2 is increased.

3.2 Forcing Early Seizure Onset

After learning how the individual variables affect the model an attempt was made to push it into early onset of a neural event. Because the x_1 variable was shown to directly affect the slope of the time scale variable z, and the x_2 variable was shown to have no effect on z (except at high magnitudes), it was hypothesized that the application of an electrical pulse could drive the epileptor into early onset of a seizure. The hypothesis was that there was a pulse whose parameters would be sufficient such that the application of the pulse would cause the slope of the z variable to become more negative, thus decreasing the time interval required to drive the system into a seizure.

A pulse was delivered to the x_1 and x_2 variables which combine to form the vertical axis of the system. Because adjusting x_1 in either direction resulted in the slope of z becoming negative it was thought that applying a positive pulse to it may force the epileptor to seizure onset. Figure 3.18 shows the results of attempting to apply a positive pulse to normal brain activity between the first and second seizures. It is shown that the application of the pulse caused the slope of z to become positive for the duration of the pulse. The slope of z resumed its negative trajectory once the pulse was discontinued. This result was not expected. Instead of decreasing the time interval, the application of a positive pulse acted to increase the duration to the onset on the next seizure.

Attempts to modify the pulse in magnitude and width were made. It proved that such modifications did not result in the early onset of a recognizable seizure. Rather, an unknown brain activity resulted. It is not known if this activity is representative of a different seizure type or if it is simply instability caused by the application of the pulse. In either case, this was not the desired result. An example of this phenomenon is shown in Figure 3.19. It was therefore determined that the application of a positive pulse did not meet the criteria required to drive the epileptor into the early onset of a seizure.

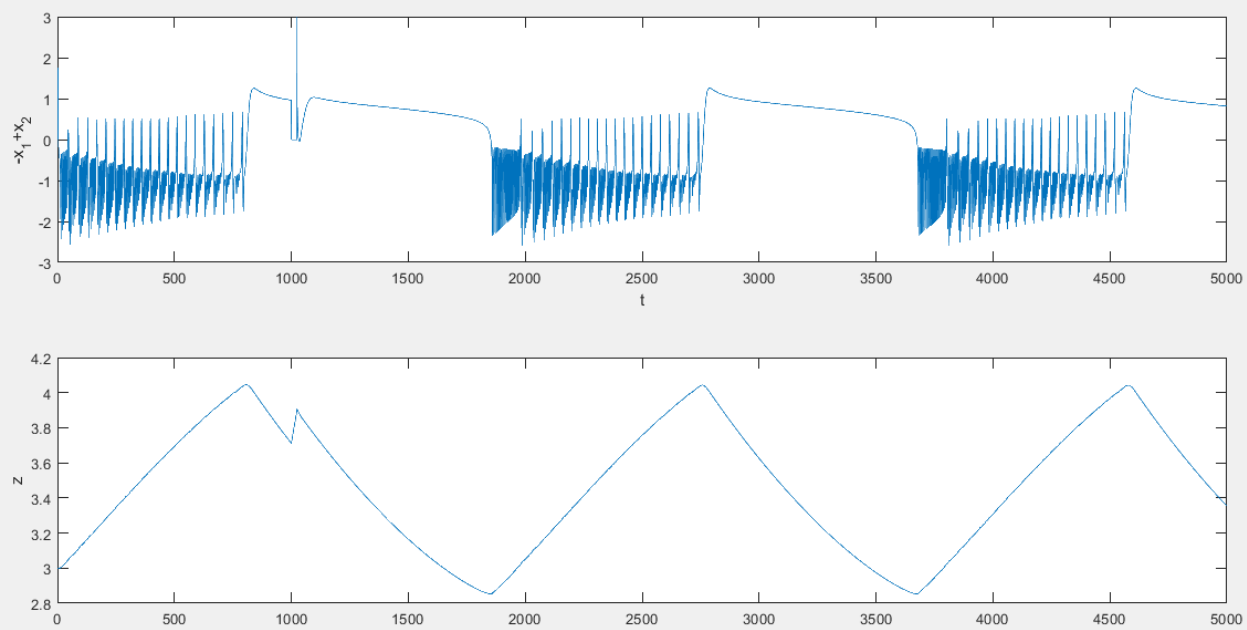

Figure 3.18: Application of a positive pulse to normal brain activity

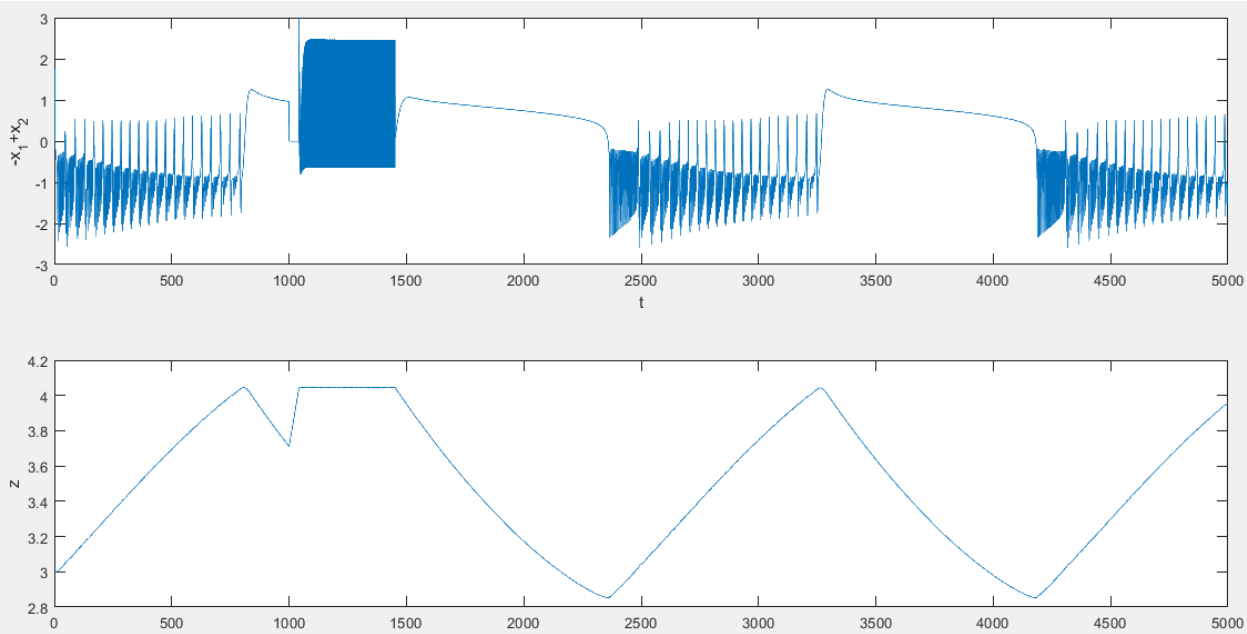

Figure 3.19: Application of a positive pulse to normal brain activity causing instability

A negative pulse was then delivered during the normal brain activity period between the first and second seizure periods. The addition of a negative pulse was found to have the desired effect on brain activity. It was shown that the application of a negative pulse caused the negative slope of z to increase in magnitude. This resulted in z reaching its minimum value rapidly thus driving the epileptor into early seizure. Figure 3.20 shows the results of delivering a pulse of amplitude $x_1 = x_2 = -5$ from $t = 1000$ to $t = 1050$. During the interval $t = 1000$ to $t = 1050$ a steep decrease in the variable z occurs. This decrease in slope reduces the time interval between seizure onset.

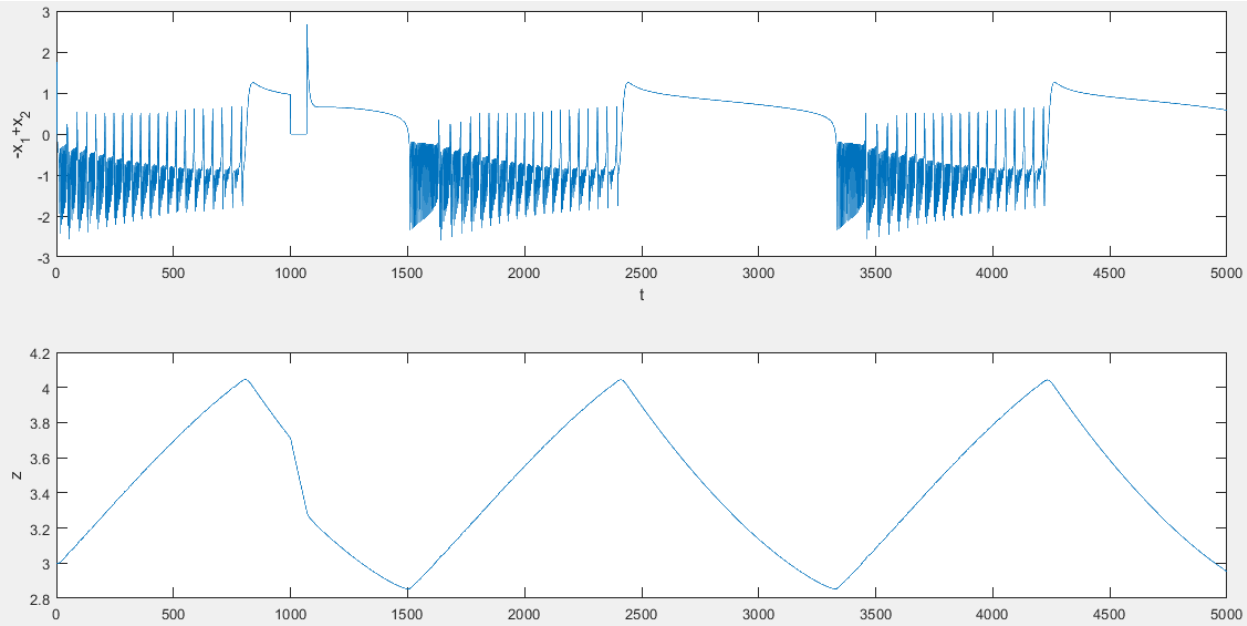

Figure 3.20: Application of a negative pulse to normal brain activity resulting in early onset seizure

These results show that it is possible to drive the epileptor into early seizure onset. This is important because, as mentioned in chapter 1, little is known about the precise mechanism that drives seizure onset. Forcing this condition in the model may aid researchers in developing an understanding of the dynamics that push normal brain activity into an abnormal state.

3.3 Early Termination of a Seizure

After learning that it was possible to force the epileptor to early seizure onset, the natural next step was to see if it was possible to terminate the abnormal neural activity by delivering an electrical pulse to the brain. Preliminary research into neurostimulation methods showed that this can be accomplished; however, it was not known if this could be reproduced in the aforementioned system.

The initial attempt was made by modifying the pulse that successfully pushed the epileptor to early seizure onset. Because the desired effect on z is to increase the positive slope, the polarity of the pulse was reversed from negative to positive. It was shown in Figure 3.18 that applying a positive pulse to x_1 and x_2 resulted in the desired effect on z. The results of delivering a pulse of amplitude $x_1 = x_2 = 5$ from $t = 2000$ to $t = 2050$ are shown if Figure 3.21. While the pulse was sufficient to terminate the seizure prematurely, it was not sufficient enough to bring z to its maximum value. Therefore, the time interval to the next seizure is shorter than normal.

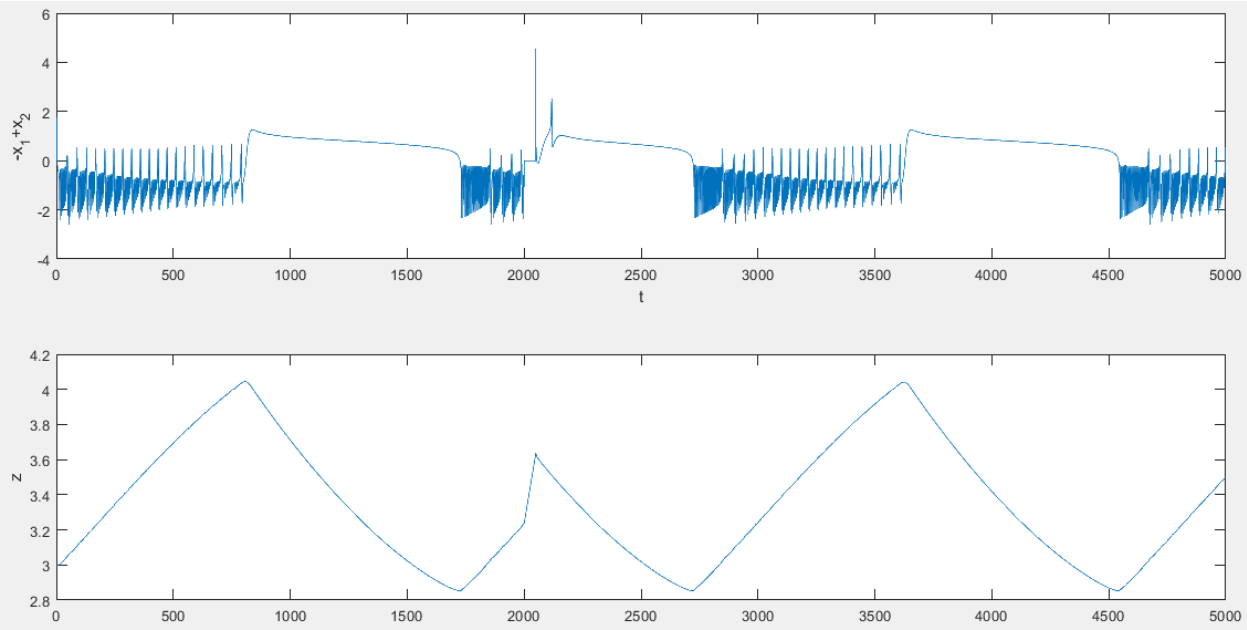

Figure 3.21: Application of a positive pulse to normal brain activity resulting in early termination of a seizure

There are two modifications that can be made to the pulse that would allow z to reach its maximum value. The first option is to increase the width of the pulse and the second is to increase the amplitude. The former is attempted first. The pulse is modified to to be delivered from $t = 2000$ to $t = 2100$. The resultant plot shown is in 3.22. The modified pulse has allowed z to reach its maximum value and has thus successfully terminated the seizure while maintaining the normal time interval to next successive onset. There are; however, some high spikes that occur when the pulse is removed at $t = 2100$. It is unknown what effect, if any, this would have on a human.

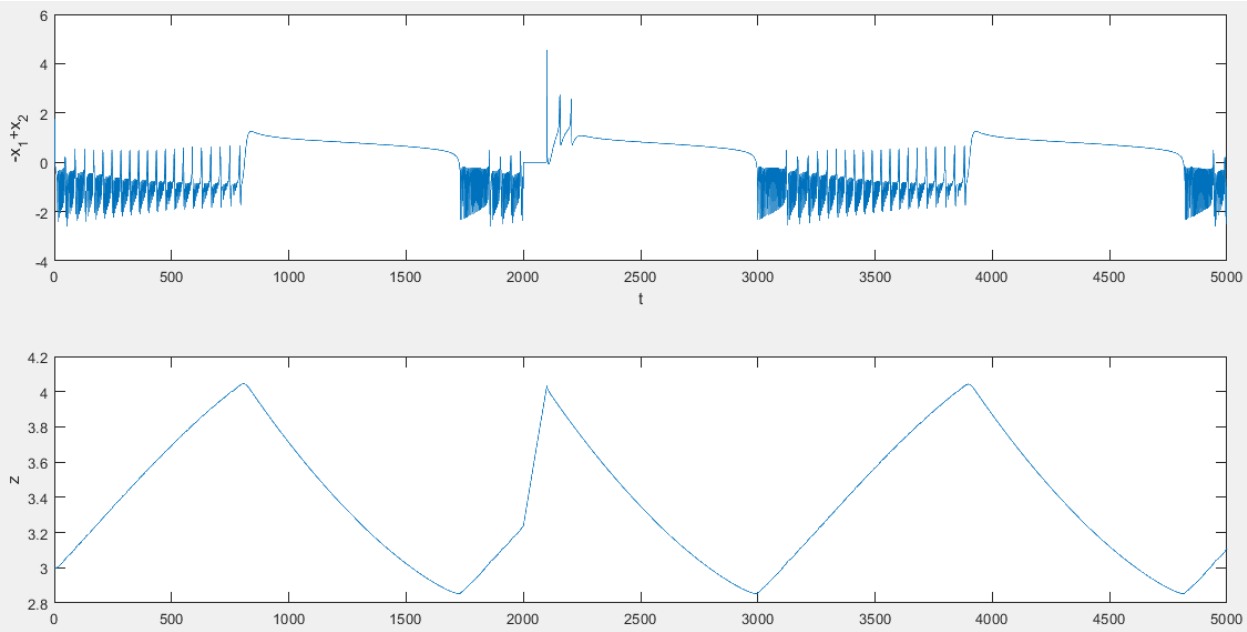

Figure 3.22: Increasing the width of a positive pulse to normal brain activity resulting in early termination of a seizure

Next, the width of the pulse is returned to its original setting. The amplitude is then increased to $x_1 = x_2 = 10.75$. The results shown in Figure 3.23 show that the pulse is sufficient to drive z to its maximum value. Additionally, the seizure is successfully terminated and the time interval to the next successive event is returned to the normal value. It is also noted that similar to the modification to pulse width, spiking occurs when the pulse is removed at $t = 2050$. Again, it is unknown what effect, if any, this would have on a human.

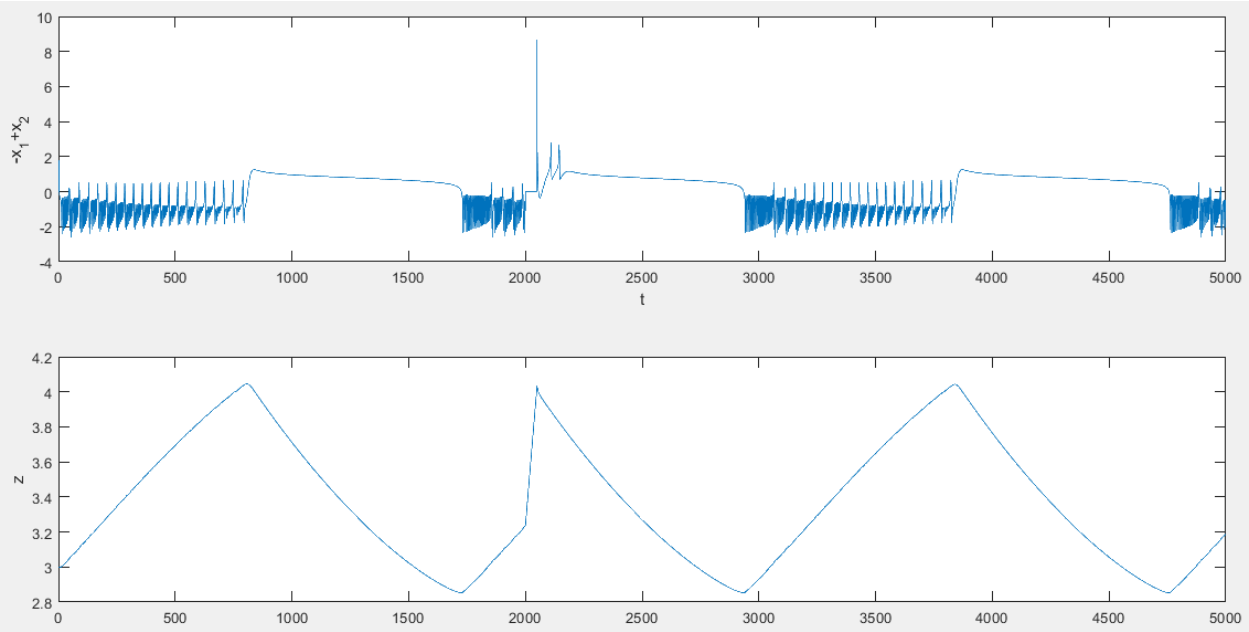

Figure 3.23: Increasing the amplitude of a positive pulse to normal brain activity resulting in early termination of a seizure

In attempt to eliminate the spike that occurs when the pulse is removed, the amplitude of the pulse is modified to be equal to the maximum value of the unmodified vertical axis. The width is then adjusted to ensure z has enough time to reach its maximum value. The resultant pulse parameters are $x_1 = x_2 = 1.256$ from $t = 2000$ to $t = 2290$. The results are shown in Figure 3.24. It is observed that the excessive spiking has been eliminated while terminating the seizure and maintaining the time interval to the next successive event.

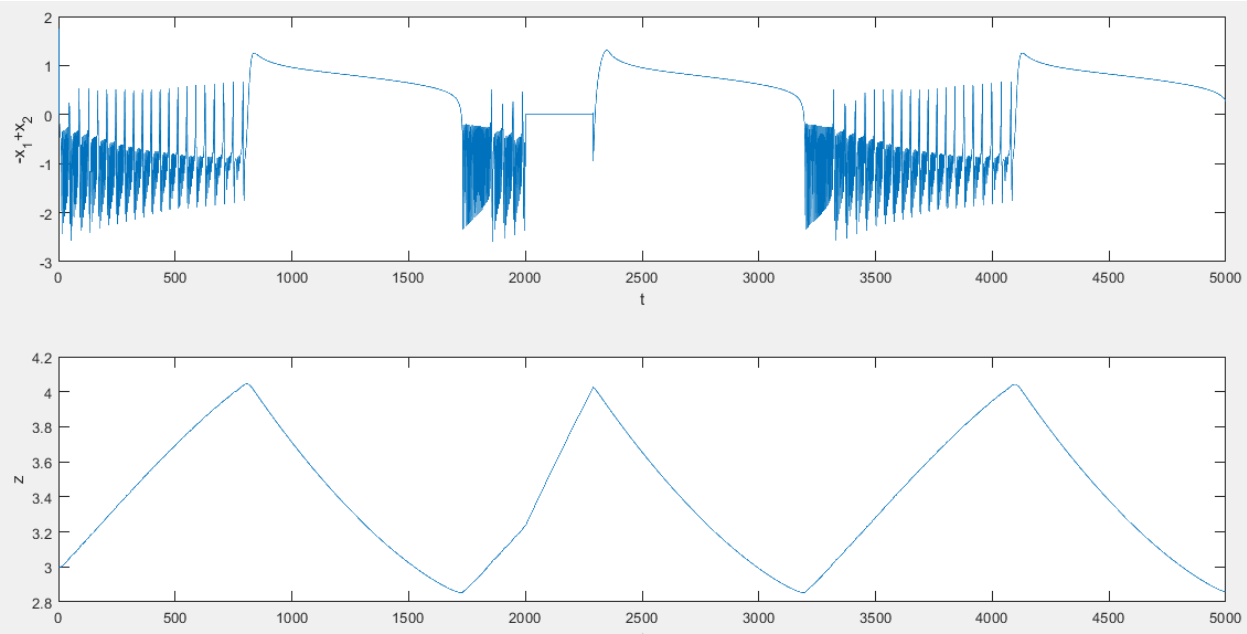

Figure 3.24: Modification of a positive pulse to prevent excessive spiking

As with pushing the epileptor to early onset seizure, it is also important to be able to terminate abnormal neural activity prematurely. It is shown that with the application of an electrical pulse it is indeed possible to cause the cessation of a seizure.

Chapter 4

Discussion and Future Work

To conclude, a discussion summarizing the work completed is presented. This discussion is followed by possible related future work.

4.1 Discussion

Epilepsy is a debilitating disease that is characterized by uncontrollable bouts of abnormal neural activity. While science knows very little with regard to the dynamics of this condition, researchers are making progress [11]. One area of progress is in the ability to model a seizure. The epileptor has been shown to be an excellent model. It has been proven to closely resemble EEG signals present in the brain during a complex partial seizure. With accurate models, researchers can further their knowledge about the dynamics of epilepsy. The models can also be used to explore possible treatment options without the need for an invasive procedure on a living subject.

A literature survey was conducted to gain an understanding as to the present state of research into this topic. Focus was placed on the understanding and recreation of the epileptor. The electrical signals were then studied to gain an understanding as to the role individual components play on the overall output. Once a firm understanding was developed with respect to individual components, the system was pushed a step further to determine if a seizure could be initiated or terminated. It was found that by delivering an electrical pulse to the model during periods of normal brain activity a seizure could indeed be initiated. The pulse was then modified and delivered to the epileptor during a seizure. It was shown that this pulse was capable of normalizing brain activity.

4.2 Future Work

The epileptor has shown to successfully model the most predominant types of seizure; however, this model is not valid for all types. Bifurcation theory, which is the basis for this paradigm, predicts a minimum of 16 different onset/offset conditions. Further development would help researchers to a more robust understanding of the dynamics driving this activity. Such models would also be beneficial to developers of treatment devices such as closed and open loop neurostimulators. With the development of an accurate model, researchers may test theoretical prototypes without the need for experimentation on live subjects. While the epileptor is a great start, a model of all seizure types would be highly desirable.

Chapter 5

Appendices

5.1 Appendix A: Matlab code

```
clc; clear all; close all

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%% Variables %%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

% Constants
X_0 = -1.6 ;
Y_0 = 1 ;
tau_0 = 2857 ;
tau_1 = 1 ;
tau_2 = 10 ;
I_rest1 = 3.1 ;
I_rest2 = 0.45 ;
gamma = 0.01 ;

% Initial Conditions
X_1 = 0 ; % 0
Y_1 = -5 ; % -5
Z = 3 ; % 3
U = 0 ; % 0
X_2 = 0 ; % 0
Y_2 = 0 ; % 0
```

```
% Set 1st element of array to initial conditions
x1(1) = X_1 ;
y1(1) = Y_1 ;
z(1) = Z ;
u(1) = U ;
x2(1) = X_2 ;
y2(1) = Y_2 ;

% Initialize while loop counters
N = 2; % array index
i = 0 ; % while counter
h = .01 ; % Runge-Kutta step size
width_of_pulse = 0; % used to define width of pulse


while (i <= 5000) % Runge - Kutta
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%% FIND RK COEFF %%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%% X1 %%%%%%%%%%%%%%%%%%%%%%%%%%
% f_11 PEICEWISE FOR 1ST R-K COEFFICIENT
if X_1 < 0
f_11 = X_1.^3 - 3 .* X_1.^2 ;
elseif X_1 >= 0
f_11 = (X_2 - 0.6 .* (Z - 4).^2) .* X_1 ;
end

k1_x1 = h .* (Y_1 - f_11 - Z + I_rest1) ; % 1ST RK COEFF.

% f_12 PEICEWISE FOR 2ND R-K COEFFICIENT
if X_1 < 0
f_12 = (X_1 + k1_x1 ./ 2).^3 - 3 .* (X_1 + k1_x1 ./ 2).^2 ;
elseif X_1 >= 0
f_12 = (X_2 - 0.6 .* (Z - 4).^2) .* (X_1 + k1_x1 ./ 2) ;
end

k2_x1 = h .* (Y_1 - f_12 - Z + I_rest1) ; % 2ND RK COEFF.

% f_13 PEICEWISE FOR 3RD R-K COEFFICIENT
if X_1 < 0
f_13 = (X_1 + k2_x1 ./ 2).^3 - 3 .* (X_1 + k2_x1 ./ 2).^2 ;
elseif X_1 >= 0
f_13 = (X_2 - 0.6 .* (Z - 4).^2) .* (X_1 + k2_x1 ./ 2) ;
```

```
end

k3_x1 = h .* (Y_1 - f_13 - Z + I_rest1) ; % 3RD RK COEFF.

% f_14 PEICEWISE FOR 4TH R-K COEFFICIENT
if X_1 < 0
f_14 = (X_1 + k3_x1).^3 - 3 .* (X_1 + k3_x1).^2 ;
elseif X_1 >= 0
f_14 = (X_2 - 0.6 .* (Z - 4).^2) .* (X_1 + k3_x1) ;
end

k4_x1 = h .* (Y_1 - f_14 - Z + I_rest1) ; % 4TH RK COEFF.

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%  Y1  %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
k1_y1 = h .* (Y_0 - 5 .* X_1.^2 - Y_1) ; % 1ST RK COEFF.

k2_y1 = h .* (Y_0 - 5 .* X_1.^2 - ...
(Y_1 + k1_y1 ./ 2)) ; % 2ND RK COEFF.

k3_y1 = h .* (Y_0 - 5 .* X_1.^2 - ...
(Y_1 + k2_y1 ./ 2)) ; % 3RD RK COEFF.

k4_y1 = h .* (Y_0 - 5 .* X_1.^2 - ...
(Y_1 + k3_y1)) ; % 4TH RK COEFF.

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%  Z  %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
k1_z = h .* (1 ./ tau_0 .* (4 .* (X_1 - X_0) ...
- Z)) ; % 1ST RK COEFF.

k2_z = h .* (1 ./ tau_0 .* (4 .* (X_1 - X_0) - ...
(Z + k1_z ./ 2))) ; % 2ND RK COEFF.

k3_z = h .* (1 ./ tau_0 .* (4 .* (X_1 - X_0) - ...
(Z + k2_z ./ 2))) ; % 3RD RK COEFF.

k4_z = h .* (1 ./ tau_0 .* (4 .* (X_1 - X_0) - ...
(Z + k3_z))) ; % 4TH RK COEFF.

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%  U  %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
k1_u = h .* (-gamma .* (U - 0.1 .* X_1)) ; % 1ST RK COEFF.

k2_u = h .* (-gamma .* ((U + k1_u ./ 2) - ...
0.1 .* X_1)) ; % 2ND RK COEFF.

k3_u = h .* (-gamma .* ((U + k2_u ./ 2) - ...
```

```
0.1 .* X_1)) ; % 3RD RK COEFF.

k4_u = h .* (-gamma .* ((U + k3_u) - 0.1 .* ...
X_1)) ; % 3RD RK COEFF.

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%  X2  %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
k1_x2 = h .* (-Y_2 + X_2 - X_2.^3 + I_rest2 + 2 .* U - .03 .* ...
(Z - 3.5)) ; % 1ST RK COEFF.

k2_x2 = h .* (-Y_2 + (X_2 + k1_x2 ./ 2) - (X_2 + k1_x2 ./ ...
2).^3 + I_rest2 + 2 .* U - .03 .* (Z - 3.5)) ; % 2ND RK COEFF.

k3_x2 = h .* (-Y_2 + (X_2 + k2_x2 ./ 2) - (X_2 + k2_x2 ./ ...
2).^3 + I_rest2 + 2 .* U - .03 .* (Z - 3.5)) ; % 3RD RK COEFF.

k4_x2 = h .* (-Y_2 + (X_2 + k3_x2) - (X_2 + k3_x2).^3 + ...
I_rest2 + 2 .* U - .03 .* (Z - 3.5)) ; % 4TH RK COEFF.

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%  Y2  %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
% f_2 PEICEWISE FOR ALL R-K COEFFICIENT
if X_2 < -0.25
f_2 = 0 ;
elseif X_2 >= -0.25
f_2 = 6 .* (X_2 + 0.25) ;
end

k1_y2 = h .* (1 ./ tau_2 .* (-Y_2 + f_2)) ; % 1ST RK COEFF.

k2_y2 = h .* (1 ./ tau_2 .* (-(Y_2 + k1_y2 ./2) + ...
f_2)) ; % 2ND RK COEFF.

k3_y2 = h .* (1 ./ tau_2 .* (-(Y_2 + k2_y2 ./2) + ...
f_2)) ; % 3RD RK COEFF.

k4_y2 = h .* (1 ./ tau_2 .* (-(Y_2 + k3_y2) + ...
f_2)) ; % 4TH RK COEFF.


%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%% NEXT ITERATION %%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

x1(N) = X_1 + (1 ./ 6) .* (k1_x1 + 2 .* k2_x1 + ...
2 .* k3_x1 + k4_x1) ;
```

```
y1(N) = Y_1 + (1 ./ 6) .* (k1_y1 + 2 .* k2_y1 + ...
2 .* k3_y1 + k4_y1) ;

z(N) = Z + (1 ./ 6) .* (k1_z + 2 .* k2_z + 2 .* ...
k3_z + k4_z) ;

u(N) = U + (1 ./ 6) .* (k1_u + 2 .* k2_u + 2 .* ...
k3_u + k4_u) ;

x2(N) = X_2 + (1 ./ 6) .* (k1_x2 + 2 .* k2_x2 + ...
2 .* k3_x2 + k4_x2) ;

y2(N) = Y_2 + (1 ./ 6) .* (k1_y2 + 2 .* k2_y2 + ...
2 .* k3_y2 + k4_y2) ;


%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%% Impulse %%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

%%% PULLS OUT OF SEIZURE - UNCOMMENT TO RUN

%  if( i >= 2000 && i < 3500 && width_of_pulse ...
%          <= 29000 && z(N) <= 4.045 )
%       x1(N) = 1.256;
%       x2(N) = 1.256;
%       width_of_pulse = width_of_pulse + 1 ;
%  end




%%% DRIVES INTO SEIZURE - UNCOMMENT TO RUN

%  if(i >= 1000 && width_of_pulse <= 5000 && ...
%              z(N) < 4.045)
%       x1(N) = -5.0;
%       x2(N) = -5.0;
%       width_of_pulse = width_of_pulse + 1 ;
%  end



%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%%%%%%%%%%%%%%%%%%%%%%%% RESET INITIAL VALUES %%%%%%%%%%%%%%%%%%
```

```
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%

X_1 = x1(N) ;
Y_1 = y1(N) ;
Z = z(N) ;
U = u(N) ;
X_2 = x2(N) ;
Y_2 = y2(N) ;

N = N + 1 ;
i = i + h;
end

t = (0:h:5000);
```

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Academic Vita

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