

**A NEW APPROACH FOR THE EPILEPSY DIAGNOSIS AND FOR THE
LOCALIZATION OF EPILEPTOGENIC BRAIN REGIONS**

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**EPİLEPSİ TEŞHİSİ VE EPİLEPTİK BEYİN
BÖLGESİNİN BELİRLENMESİ İÇİN YENİ BİR
YAKLAŞIM**

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PREFACE

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ABBREVIATIONS

L	: lag operator
Δ	: first-difference operator
$\otimes$	: Kronecker product
$(x y)$	: x conditional on y
ADF	: augmented Dickey-Fuller
AR	: autoregression
ARIMA	: autoregression integrated moving average
ARMA	: autoregressive moving average
BM	: Brownian motion
CI(d,b)	: Co-integrated of order d,b
DF	: Dickey-Fuller
DGP	: Data generation process
ECM	: Error correction mechanism
iid	: Independently and identically distributed
OLS	: ordinary least squares
SC	: Schwarz information criterion
SD	: Standard deviation
SE	: Standard error
VAR	: Vector autoregression
W(r)	: Wiener process with increments of variance r

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EPİLEPSİ TEŞHİSİ VE EPİLEPTİK BEYİN BÖLGESİNİN BELİRLENMESİ İÇİN YENİ BİR YAKLAŞIM

ÖZET

Bu çalışmamda temel olarak, insan beyninden alınan EEG işaretlerinin doğasını anlamak üzerine çalıştım. Yürüttüğüm çalışmanın ana eksenini Alzheimer hastası olduğundan kuşkuilanılan bir grup hastanın ve ayrıca Sara hastası olduğundan kuşkuilanılan başka grup bireylerin 19 kanaldan 76000 örnek olarak kaydedilmiş EEG işaretlerinin Zaman Serileri Analizi yapmak teşkil etmektedir.

Bilindiği üzere Zaman Serisi Analizleri, ağırlıklı olarak Makroekonomi, Ekonometri ve Finansal Analiz bilim dalları tarafından kullanılmaktadır; ilgilenilen değişkenlerin gelecekte alacağı değerleri tahmin etmek üzerine yoğunlaşmıştır. Beyin EEG işaretlerini araştıran Biomedikal ilmine veya Tıp ilimlerine ait çalışmaların ortak yaklaşımları uzun senelerden beri söz konusu işaretlere frekans domeni analizi yapmak, onların neticelerini değerlendirmek ve görüntüleme çalışmaları üzerinde durmak yönünde olmuştur. Bu tip bir metodik yönelimin nedenini, Beyin EEG’inde “değişmez” özellikler gösteren “patern” arama arzusu olarak nitelendirebiliriz; tahmin edilebileceği üzerine bu yaklaşım Kalp işaretinin değişmez bir patern’i haiz olmasından kaynaklanmaktadır. Ne var ki, söz konusu geniş literatür tarandığında, Beyin EEG’sinin çözümü konusunda pek fazla yol alınamadığı görülür. Hatta öyle ki, görüntüleme tekniklerine istinaden, özellikle çok sık Sara krizi geçiren hastalara beyinlerinin “anormal” işaret ürettiği varsayılan bölgesine cerrahi müdahale yapılmak suretiyle gerçekleştiren tedaviler dahi bazen boşboşuna olmaktan öteye geçememekte ve krizler nüksetmektedir. Son görüntüleme tekniklerinin kullanılıyor olması tıbbi bağışlanır kılar mı, üzerinde durmak gerekir.

Bu noksanın ayırdımında olan bazı araştırmacılar geçtiğimiz beş yıldan başlayarak, EEG işaretlerine frekans domeni analizi yaparak patern aramak yerine, mevcut görüntüleme tekniklerini Zaman Serisi Analizi ile gerçekleştirme çalışmalarına başlamışlardır.

Biz çalışmamızda Beyin EEG işaretlerine Zaman Serisi Analizi uygulayacağımızdan mütevellit, Biyomedikal çalışmaların bu kısım ile alakalı olan bölümü üzerinde duracağız. Son yıllarda, frekans domeni analizlerinin eksikliklerinden ötürü ortaya çıkmış ve görüntüleme ve Beyin haritalaması açısından daha verimli olduklarını iddia eden bu zaman-uzay düzlemli modelleri inceleyerek, varsa onların “fizyolojik” ve “nörolojik” açıdan makul olmayan varsayımlarını araştırarak ve kendi orijinal inceleme metodumuzu kuracağız. Bu tip bir inceleme metodunun faydalı olacağını düşünüyoruz, zira her “yeni” daha öncekilerinin noksanlıklarını kapattığını iddia

ederek vukuu bulur ve eğer biz bu “yeni” lerin noksanlıklarını ortaya çıkarabilirsek, çalışmamızın doğruluğunu en azından bir bacadan test etmiş olabiliriz. Gerçekleştirdiğimiz Zaman Serisi Analizi’nin özünü şu yaklaşımımız oluşturmaktadır: Acaba Beyin EEG işaretleri rastgele üretilmekte olan işaretler olamaz mı?

Dolayısıyla, teknik olarak, hem “normal” olduğu düşünülen insandan alınan Beyin EEG sinde, hem Alzheimer olduğundan kuşku edilen insandan alınan EEG’de ve hem de Sara hastası olduğundan şüphelenilen bireylerden alınan Beyin EEG’lerinde, dataların “*durağan süreçler*” mi, “*durağan olmayan süreçler*” mi izlediğini araştıracağız. Eğer iki tip hastalıktan birinde veya ikisinde birden *normaldekinden farklı bir durumla* karşılaşsak bunun nedenlerini aramaya girişeceğiz. Buna paralel olarak ortaya çıkacak fizyolojik ve nörolojik bazı suallerin yanıtlarını arayacağız.

Bulgularımız ışığında da, son beş yıldır gerçekleştirilen görüntüleme tekniğindeki yeniliklerin eksiklerini sergileyeceğiz. Daha öncekilerin eksiklerini zaten söz konusu “yeni” ler sergilemektedir.

Bulgularımızı en kaba hatları ile şu şekilde sıralayabiliriz:

1. Normal insana ait Beyin EEG işareti hem çok büyük örneklerde (uzun süreçte) hem de bu örneklerle ait alt örnek aralıklarında (alt süreçlerde) *durağan* olarak tespit edilmiştir.
2. Alzheimer olduğundan kuşku edilen hastalarda da Zaman Serisi Analizi açısından, normal bireyinkinden farklı hiçbir duruma rastlanılmamıştır.
3. Fakat, Sara olduğundan kuşku edilen hastalarda durum tamamen farklıdır. Çok büyük gözlem aralığında, işaret *durağan* karakter sergiliyor olsa da, küçük gözlem aralıklarında işaret *durağan olmayan* süreç takip etmektedir. Buna paralel olarak *durağan olmayan* işaret üreten bölgeler arasında *ortak hareket* mevcuttur. Yani, bir bölge bir anda *durağan olmayan* data üretmeye başlamakta ve bu durum çok küçük zaman aralığında diğer bölgeleri tetiklemekte ve söz konusu zaman aralığından başlayarak *durağan olmayan süreç*’in tüm beyne yayılmasına neden olmaktadır. Bu bulgu bile tek başına, Sara literatüründe ancak son günlerde ortaya atılan yeni bir iddiaya işaret etmesi açısından değerlidir. Sara krizleri kesinlikle tek kaynaktan başlamakta ve yayılmaktadır.

Kabaca 3. nolu maddede açıklanan bulgumuzun üzerine giderek ve “krizi önceden tahmin edebilir miyiz?” veya “Sarı hastalar için daha gerçekçi bir görüntüleme metodu oluşturabilir miyiz?” sorularından başlayarak bir dizi fizyolojik ve nörolojik soruyu yanıtlamaya çalışacağız. Tekrar ifade etmekte fayda görüyoruz. Böyle bir çalışma henüz ne Biyomedikal literatüründe ne de Tıp literatüründe yapılmamıştır; dolayısıyla neticelerimiz ilk kez bu tez vasıtası ile ortaya konmaktadır. Umuyoruz ki, Sarı hastalar için yeni bir gelişme olarak kayda geçer.

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SUMMARY

In this study, I examined the nature of Brain EEG data by using Time Series Analysis methods. As we know, Time Series Analysis generally used in Economics and in its branches Macroeconomics, Econometrics and Financial Analysis to model the behaviour and to estimate the values of some interested variables. Mainly, we are interested in the structure of the data generated by unobserved sources in human brain.

We begin our analysis by examining the main tools of Time Series Analysis branch of Brain EEG research, and by handling very new approaches from the literature, we figure out the usefulness of existing methods for both modelling and imaging power or brain mapping, and finally we derive the “ill-posed” sides of these very new studies while comparing them with already existing methods. We find this study route efficient because we know that every “new” approach or method claims that its reason of occurring depends on the “ill-posed” sides of existing ones. By the way, if we explore and analyze the newest researches and if we can success to figure out the “ill-posed” and “unplausible” and “counterintuitive” sides of these newest methods, then we can reach more accurate results for our objective problem.

The application side of this study: we analyze the EEG recordings of certain individuals that are under suspect of some mental illnesses like Alzheimer or Epilepsy. In the last part of our study we briefly and theoretically explain what Non-stationary data is, how can it be inferred from special statistical tests, what their estimation methods are. Hence we apply these theories to the EEG recordings of both Epilepsy and Alzheimer. To the best of our knowledge there is not any similar research or research idea in the related Brain EEG modelling and search literature.

In general frame, our results are:

1. We find that the recordings of 30 individuals from this group contains some sample subintervals in which the observed data has Non – Stationary character. According to best of our knowledge, this means that: All of these individuals have Epilepsy Disease. The Non – Stationarity in their data shows us the temporary effects of Epilepsy.
2. As we examined the data of one individual taken from 19 sources or region seperately, we found that in our every test interval the Non – Stationarity exists

within some of these 19 different signals. This amount also varies from one individual to another individual. We think that the reason is clear and occurs from the physiological differences among individuals.

3. However, there is a common discovery for all Epilepsy - diseased people. The signal sources or regions that generate Non- Stationary data have co-movements with its neighbours because, the Non-Stationary data generated by them have Co-Integration. This charecteristic behaviour is observed in recordings of all individuals. This strictly shows us that: Epilepsy is generated in only one region or from only one signal source in Brain, and after, it is diffused to other regions within miliseconds.

Moreover, we found how can we estimate the region that triggers Epilepsy Crise.

INTRODUCTION

Our study examines a highly debated subject in Brain Research within a framework of special application of Time - Series analysis. Since the invention of EEG recording techniques, researchers in Biomedical area have tried to develop better imaging methods to contribute medical studies on Brain research. In general, Biomedical research area of Brain mainly concerns with frequency domain analysis of certain signals and seeks for some *patterns* that “make sense” to explain some invariant links between them and the events occurring within Brain. This approach can be viewed to search for a *reference system* that may distinguish normal individuals from individuals that suffer from some mental illnesses.

In brain EEG research literature there exists many different mathematical modellings and imaging techniques based on them. We can mainly group these studies in two parts: Frequency domain analysis and Time – Series analysis. Even if the first part has been performed for years, the second group has been occurred essentially within last decade. One of the most important reason for its being late may be that: The original science branch and “main engine” of Time – Series Analysis are Economics and Econometrics. Although this multi disciplinary handicap exists, some neuroscientists have found its worthwhile to examine Brain EEG recordings in the context of Time – Series Analysis especially for five years. Their main aspects are viewed as follows: Assuming some unobserved signal sources in gray-matter of Brain and analyzing EEG recordings as observed signals generated by these sources and finally trying to estimate the location of these unobserved sources within Brain. We can easily see that, this effort is even deriving from their willingness to “imaging” or brain mapping. Hence these analysis are mainly grouped under the title “dynamical inverse problems”.

In this study we define a route within this “huge” research literature and try to reach useful results and arguments. As a starting point we take Time – Series Analysis contributions of Brain EEG research and by following the route of the researchers occupying with this research title, we both explore their studies, criticise them and try to approach the problem in a different way to better understand the nature of Brain EEG. So we begin our analysis by examining the main tools of Time –Series Analysis branch of Brain EEG research, and by handling very new approaches from the literature, we figure out the usefulness of existing methods for both modelling and imaging power or brain mapping, and finally we derive the “ill-posed” sides of these very new studies while comparing them with already existing methods. We find this study route efficient because we know that every “new” approach or method claims that its reason of occuring depends on the “ill-posed” sides of existing ones. By the way, if we explore and analyze the newest researches and if we can success to figure out the “ill-posed” and “unplausible” and “counterintuitive” sides of these newest methods, then we can reach more accurate results for our objective problem.

The ordering of our study as follows: In the first part, the EEG analysis methods used in biomedical literature are examined; moreover, following from the newest studies concerning Time Series Analysis in the Brain EEG research literature, we analyze *Spatiotemporal Model* and its estimation method *Spatiotemporal Kalman Filtering* which has been introduced by certain neuroeconomists and physiciens to better model unobserved sources (states) in Brain. After we examine this new extension and crtisize its “strong assumptions”, it is taken into account one of the most interesting solutions to these “unplausible” assumptions of Spatiotemporal modelling of unobserved signal sources of Brain by introducing Non-Stationarity of the shocks within ARCH/GARCH processes framework, the detailed explanation of which is given in the Appendix. Behind this analysis, the notion of “Non-Stationarity” in this part will be a convenient base for our original analysis that will take place in fourth part.

Finally, in the second part of this study, we analyze the EEG recordings of certain individuals that are under suspect of some mental illnesses like Alzheimer or Epilepsy. In the last part of our study we briefly and theoretically explain what Non-stationary data

is, how can it be inferred from special statistical tests, what their estimation methods are. Hence we apply these theories to the EEG recordings of both Epilepsy and Alzheimer. To the best of our knowledge there is not any similar research or research idea in the related Brain EEG modelling and search literature.

In the second part of our study we especially try to find the answers of such questions cited below: We focus on Epilepsy and Alzheimer EEG recordings each of which contains 76000 samples from 19 different regions (signal sources) of Brains of various individuals some of whom are under suspect of being Epilepsy and some of whom are under suspect of being Alzheimer.

1. We search first, Non – Stationarity in whole sample string; second, we explore Non – Stationarity within different subintervals of this whole recording of every region one by one. Intuitively we know that the whole recording (76000 sample) data can not be Non – Stationary. The reason is clear and physiological: The observed effects of these mental illnesses *must have been temporary*. So, we will explicitly search different subintervals and if we find such a subinterval within whole recording in which the data is Non – Stationary, then we have to explain why in following subintervals the data changes its nature and becomes Stationary? What is the reason and meaning of this behaviour change both physiologically and neurologically?
2. Depending upon the response of first question above, if it is found that, the datas generated by different regions of Brain have Non – Stationary behaviours within some subintervals, what does it mean physiologically? Secondly, is there a co-movement between these Non – Stationary datas which are generated by neighbourhood sources or regions in Brain? If there exists such a co-movement relation (*Co-Integration*) between the Non –Stationary datas generated by any two or more regions, does this mean that one of these sources triggers the Non – Stationary data generation by neighbour regions and hence causes its diffusion to the whole Brain?
3. Thirdly, if there exists a pioneer unobserved signal source triggering the generation of Non- Stationary data, can we estimate its location within Brain? Hence could we estimate the Epilepsy crises?

Moreover, one can think that why we need to explain Spatiotemporal Models in our thesis. In the part 2.3. we will compare our findings with the fundamental assumptions of newest techniques (Spatiotemporal Models) and criticise the *imaging* and *Brain Mapping* techniques using these type of models.

In this study we examined the nature of Brain EEG taken from various individuals either normal or suffering from a mental disease. For this real data analysis I used the clinical EEG data taken from various (30) individuals under the suspect of Epilepsy, (4) individuals under the suspect of Alzheimer. The number of measurements given with the standard 10–20 system of clinical EEG recordings. These datas can be supplied upon to request.

1. EEG DATA ANALYSIS AND EEG REGENERATION METHODS IN BIOMEDICAL ENGINEERING

The EEG (electroencephalograph) measures brainwaves of different frequencies within the brain. Electrodes are placed on specific sites on the scalp to detect and record the electrical impulses within the brain. A frequency is the number of times a wave repeats itself within a second. It can be compared to the frequencies that you tune into on your radio. If any of these frequencies are deficient, excessive, or difficult to access, our mental performance can suffer. Amplitude represents the *power* of electrical impulses generated by brain. Volume or intensity of brain wave activity is measured in microvolts. The raw EEG has usually been described in terms of frequency bands: Gamma greater than 30(Hz) BETA (13-30Hz), ALPHA (8-12 Hz), THETA (4-8 Hz), and DELTA(less than 4 Hz).

Brain Wave Frequencies, Delta (0.1 to 3 Hz):

The lowest frequencies are delta. These are less than 4 Hz and occur in deep sleep and in some abnormal processes also during experiences of "empathy state". Delta waves are involved with our ability to integrate and let go. It reflects unconscious mind. It is the dominant rhythm in infants up to one year of age and it is present in stages 3 and 4 of sleep. It tends to be the highest in amplitude and the slowest waves. We *increase* Delta waves in order to decrease our awareness of the physical world. We also access information in our unconscious mind through Delta. Peak performers *decrease* Delta waves when high focus and peak performance are required.

However, most individuals diagnosed with Attention Deficit Disorder, naturally *increase* rather than decrease Delta activity when trying to focus. The inappropriate Delta response often severely restricts the ability to focus and maintain attention. It is as if the brain is locked into a perpetual drowsy state.

Another way to look at Delta is to imagine you are driving in a car and you shift into 1st gear....you're not going to get anywhere very fast. So Delta would represent 1st gear.

Distribution: generally broad or diffused may be bilateral, widespread

Subjective feeling states: deep, dreamless sleep, non-REM sleep, trance, unconscious

Associated tasks & behaviors: lethargic, not moving, not attentive

Physiological correlates: not moving, low-level of arousal

Effects of training: can induce drowsiness, trance, deeply relaxed states

Theta (4-8 Hz):

The next brainwave is theta. Theta activity has a frequency of 3.5 to 7.5 Hz and is classed as "slow" activity. It is seen in connection with creativity, intuition, daydreaming, and fantasizing and is a repository for memories, emotions, and sensations. Theta waves are strong during internal focus, meditation, prayer, and spiritual awareness. It reflects the state between wakefulness and sleep. It relates to subconscious. It is abnormal in awoken adults but is perfectly normal in children up to 13 years old. It is also normal during sleep. Theta is believed to reflect activity from the limbic system and hippocampal regions. Theta is observed in anxiety, behavioral activation and behavioral inhibition. When the theta rhythm appears to function normally it mediates and/or promotes adaptive, complex behaviors such as learning and memory. Under unusual emotional circumstances, such as stress or disease states, there may be an imbalance of three major transmitter systems, which results in aberrant behavior. Back to our car example, Theta would be considered 2nd gear. Not as slow as 1st gear (Delta) but still not very fast.

Distribution: usually regional, may involve many lobes, can be lateralized or diffuse;

Subjective feeling states: intuitive, creative, recall, fantasy, imagery, creative, dreamlike, switching thoughts, drowsy; "oneness", "knowing"

Associated tasks & behaviors: creative, intuitive; but may also be distracted, unfocused

Physiological correlates: healing, integration of mind/body

Effects of Training: if enhanced, can induce drifting, trance-like state. If suppressed, can improve concentration, ability to focus attention

Alpha (8-12 Hz):

Alpha waves are those between 7.5 and 13(Hz). Alpha waves will peak around 10Hz. Good healthy alpha production promotes mental resourcefulness, aids in the ability to mentally coordinate, and enhances overall sense of relaxation and fatigue. In this state you can move quickly and efficiently to accomplish whatever task is at hand. When Alpha predominates most people feel at ease and calm. Alpha appears to bridge the conscious to the subconscious. It is the major rhythm seen in normal relaxed adults - it is present during most of life especially beyond the thirteenth year when it dominates the resting tracing. Alpha rhythms are reported to be derived from the white matter of the brain. The white matter can be considered the part of the brain that connects all parts with each other. Alpha is a common state for the brain and occurs whenever a person is alert (it is a marker for alertness and sleep), but not actively processing information. They are strongest over the occipital (back of the head) cortex and also over frontal cortex. Alpha has been linked to extroversion (introverts show less), creativity (creative subjects show alpha when listening and coming to a solution for creative problems), and mental work. When your alpha is within normal ranges we tend to also experience good moods, see the world truthfully, and have a sense of calmness. Alpha is one of the brain's most important frequencies to learn and use information taught in the classroom and on the job. You can increase alpha by closing your eyes or deep breathing or decrease alpha by thinking or calculating. Alpha-Theta training can create an increase in sensation, abstract thinking and self-control. In our car scenario, Alpha would represent neutral or idle. Alpha allows us to shift easily from one task to another.

Distribution: regional, usually involves entire lobe; strong occipital w/eyes closed

Subjective feeling states: relaxed, not agitated, but not drowsy; tranquil, conscious

Associated tasks & behaviors: meditation, no action

Physiological correlates: relaxed, healing

Effects of Training: can produce relaxation

Sub band low alpha: 8-10: inner-awareness of self, mind/body integration, balance

Sub band high alpha: 10-12: centering, healing, mind/body connection

Beta (above 12 Hz):

Beta activity is 'fast' activity. It has a frequency of 14 and greater Hz. It reflects desynchronized active brain tissue. It is usually seen on both sides in symmetrical distribution and is most evident frontally. It may be absent or reduced in areas of cortical damage. It is generally regarded as a normal rhythm and is the dominant rhythm in those who are alert or anxious or who have their eyes open. It is the state that most of brain is in when we have our eyes open and are listening and thinking during analytical problem solving, judgment, decision making, processing information about the world around us. Beta would represent overdrive or hyperdrive in our car scenario. The beta band has a relatively large range, and has been divided into low, midrange and high.

Low Beta (12-15 Hz), formerly "SMR":

Distribution: localized by side and by lobe (frontal, occipital, etc)

Subjective feeling states: relaxed yet focused, integrated

Associated tasks & behaviors: low SMR can reflect "ADD", lack of focused attention

Physiological correlates: is inhibited by motion; restraining body may increase SMR

Effects of Training: increasing SMR can produce relaxed focus, improved attentive abilities.

Midrange Beta (15-18 Hz)

Distribution: localized, over various areas. It may be focused on one electrode.

Subjective feeling states: thinking, aware of self & surroundings

Associated tasks & behaviors: mental activity

Physiological correlates: alert, active, but not agitated

Effects of Training: can increase mental ability, focus, alertness, IQ

High Beta (above 18 Hz):

Distribution: localized, may be much focused.

Subjective feeling states: alertness, agitation

Associated tasks & behaviors: mental activity, e.g. math, planning, etc.

Physiological correlates: general activation of mind & body functions.

Effects of Training: can induce alertness, but may also produce agitation, etc.

Gamma (above 36 Hz):

Gamma is measured between 36-44 (Hz) and is the only frequency group found in every part of the brain. When the brain needs to simultaneously process information from different areas, it's hypothesized that the 40Hz activity consolidates the required areas for simultaneous processing. A good memory is associated with well-regulated and efficient 40Hz activity, whereas a 40Hz deficiency creates learning disabilities.

Gamma (40 Hz):

Distribution: very localized

Subjective feeling states: thinking; integrated thoughts

Associated tasks & behaviors: high-level information processing, "binding"

Physiological correlates: associated with information-rich task processing

Effects of Training: not known

The EEG (electroencephalogram) displays the electrical activity of the brain. Nerve cells in the brain are constantly creating very small electrical signals, whether a patient is waking or sleeping. The EEG machine contains amplifiers which make these signals, or brainwaves, big enough so we can see them. The electrical signals are picked up by electrodes glued to the scalp, and travel to the amplifiers of the EEG machine and then are either written out on paper or saved on the hard drive of a computer and displayed on the computer's monitor. There are two electrodes plugged into each amplifier on the EEG machine. The amplifier looks at the two electrode signals coming into it and cancels out signals that are the same. So, the signal that you see on the paper or on the computer screen is actually the difference between the electrical activity picked up by two electrodes. The placement of the electrodes is important because the closer the electrodes are to each other the less differences in their brainwaves. One may see the

locations of electrodes and related brain regions in Figure 1. Therefore, if the electrodes are too close, the EEG will look like a straight line instead of showing the brainwaves. This is why the technician measures the head of each patient. Measuring the electrode placements allows the technician to have equal distances between all the electrodes to get clear and symmetrical brainwaves. The technician also has to constantly watch the EEG to make sure the electrodes are working properly and to eliminate any artifact or electrical interference that might occur. The EEG is still the leading test used to help diagnose seizures. Many people do not have a detectable brain lesion causing their seizures, and tests like the MRI and CT scan show normal brain structure. The EEG, however, can show abnormal electrical function of the brain even when these other tests are normal. In summary, the EEG is an important test used in the treatment of epilepsy because it shows abnormal brain signals even if the brain structure is normal. The EEG records the electrical activity of the brain between seizures, and during seizures. By looking at the patterns of the EEG in these different stages the doctor can tell if the patient has seizures, which type of epilepsy the person has, and which part of the brain is causing the seizures. This information can help the doctor to determine which medication is appropriate to treat the seizures. If medications do not help, and a specific area was found to cause the seizures, then the person may be eligible for neurosurgical treatment.

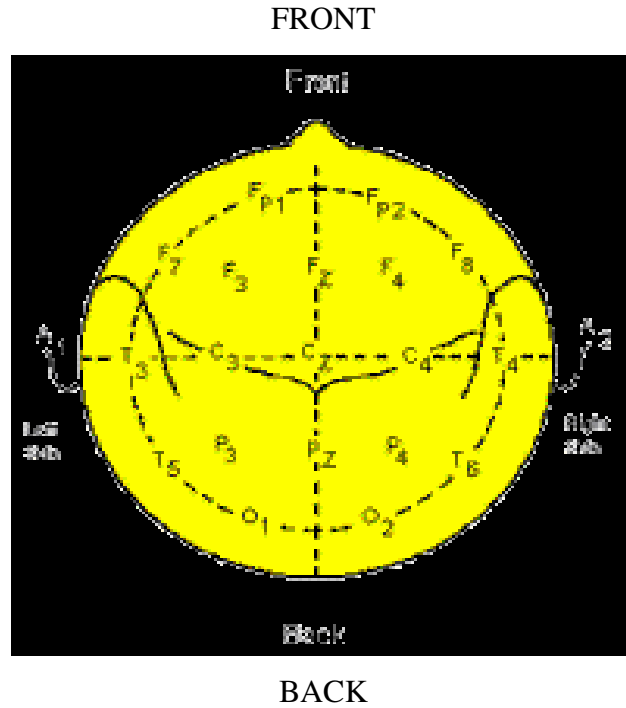


Figure 1: The Locations of Scalp Electrodes

Recordings of electromagnetic fields emanating from human brain are well known to provide an important source of information about brain dynamics. Electrical potentials on the scalp surface are very easy to measure at a set of electrodes attached to the skin; as a result, multivariate electroencephalographic (EEG) time series are obtained. With considerably higher technical effort, magnetoencephalographic (MEG) time series can also be recorded. It is by now widely accepted that the sources of these electromagnetic fields are electrical currents within networks of neurons in the cortex and other gray matter structures of brain; while part of this current remains confined within the dendritic trunks (primary currents), another part flows through the extracellular volume (secondary currents) (Nunez,1981). To obtain more direct access to the dynamics governing the activity of these networks of neurons, it would be desirable to have direct estimates of these sources. The estimation of these sources from recordings of EEG or MEG has recently become a subject of intense research (Darvas et al., 2001), (Gorodnitsky et al., 1995), (Grave de Peralta Menendez and Gonzalez Andino, 1999),

(Greenblatt, 1993), (Pascual-Marqui et al., 1994), (Phillips et al., 2002), (Riera et al., 1998), (Scherg and Ebersole, 1994), (Schmitt et al., 2002), (Yamashita et al., 2004), for a recent review see (Baillet et al., 2001). Although we examine in this study the estimation methods that for EEG, but the ideas and methods to be presented remain equally valid for the MEG. In general two main classes of source models have been developed: “equivalent current dipole” approaches (also known as “parametric” methods), in which the sources are modeled by a relatively small number of focal sources at locations to be estimated from the data, and “linear distributed” approaches (also known as “imaging” or “current density reconstruction” methods), in which the sources are modeled by a dense set of dipoles distributed at fixed locations (which, in analogy to the case of magnetic resonance imaging, we shall call “voxels”) throughout the head volume. Examples of parametric approaches include least-squares source estimation Scherg and Ebersole (1994) and spatial filters, such as beamforming and multiple signal classification (“MUSIC”) approaches (Mosher et al., 1992). We will explore and compare the linearly distributed model approach and its efficiency. It is a characteristic problem of distributed source models that a large number of unknown quantities has to be estimated from a much smaller number of measurements; because of this, we are facing a problem that does not possess a unique solution, known as “*inverse problem*”. The number of measurements given at one instant of time may be as low as 18, if the standard 10–20 system of clinical EEG recordings is employed; by increasing the number of electrodes, we may eventually obtain up to a few hundred measurements, but they will fail to provide an equivalent amount of independent information due to strong correlations between adjacent electrodes. On the other hand, the number of voxels will typically be several thousand, and furthermore at each voxel site a full three-dimensional current vector has to be modelled. To identify a unique solution (i.e., an “inverse solution”), additional information has to be employed. So far this has been done mainly by imposing constraints on the inverse solution. Certain constraints can be obtained from neurophysiology Phillips et al., (2002); as an example, it is reasonable to assume that only voxels within gray matter contribute substantially to the generation of the electromagnetic fields; other constraints refer to the probable direction of local

current vectors at specific locations. But such constraints do not suffice to remove the ambiguity of the inverse solution. For this purpose, much more restrictive constraints are needed, such as the minimum-norm constraint suggested by Hamalainen and Ilmoniemi (1984) or the maximum-smoothness constraint suggested by (Pascual-Marqui et al., 1994). These constraints can be applied independently for each instant of time, without accessing the data measured at other instants of time; therefore, we will say that the resulting inverse solutions represent solutions of the “*instantaneous*” *inverse problem*. The idea of including data from more than a single instant of time into the estimation of inverse solutions is attractive, since more information becomes available for the solution of an ill-posed problem; consequently, there has recently been growing interest in generalising the instantaneous inverse problem to “dynamical” inverse problems and to develop algorithms for its solution (Darvas et al., 2001; Kaipio et al., 1999; Schmitt et al., 2002; Somersalo et al., 2003).

In this part of the study, we will examine the newest method (Galka 2004) that develops a new interpretation of the dynamic inverse problem in its most general shape, and that proposes a new approach to its solution within this “dynamic inverse problem” and we trace related literature by handling also the older studies and comparing their solutions. In contrast to most previous work, the study of Galka et al., (2004) does not approach this problem within a constrained least squares (or, equivalently, Bayesian) framework, but by reformulating it as a spatiotemporal state space filtering problem. So far, the dynamical aspect of the available algorithms was essentially limited to imposing temporal smoothness constraints Baillet and Garnero (1997), Schmitt et al., (2002); from a time-domain modelling perspective, such constraints correspond to the very special case of a spatially noncoupled random walk model (Somersalo et al., 2003). By appropriate generalisation, their approach will permit the use of much more general predictive models in this context, such that a consistent description of the spatiotemporal dynamics of brain becomes possible. It should be stressed that general predictive models can also be incorporated into the framework of constrained least squares, and in a companion to this paper we will discuss in more detail the application of this idea to the inverse problem of EEG generation (Yamashita et al., 2004).

As the main tool for task of Galka 2004, they adapt the well-known Kalman filter Chui and Chen (1999) to spatiotemporal filtering problems; it becomes evident that Kalman filtering provides a natural framework for addressing the dynamical inverse problem of EEG generation. Theoretically, by employing a very high-dimensional state vector, standard Kalman filtering could deal with any spatiotemporal filtering problem, but the specific nature of the spatial dimensions (such as neighbourhood relationships between voxels) would not be properly captured, and computational expenses would soon become prohibitively large. By assuming a properly chosen structure of the dynamics and certain additional approximations, the intractable high-dimensional filtering problem can be decomposed into a coupled set of tractable low-dimensional filtering problems. This adaptation can be regarded as a generalisation of standard Kalman filtering to the case of partial (space–time) differential equations. From system theory, it is known that the sufficient condition for successful application of Kalman filtering is observability of the given state space system, as represented by its state transition parameter matrix (or, in the nonlinear case, the corresponding Jacobian matrix) and its observation matrix (Kailath, 1980; Kalman et al., 1969). Although they did not rigorously prove observability, they discussed the application of this concept to the state – space model and demonstrated through an explicit numerical simulation study that Kalman filtering can successfully be applied. From this simulation, it also becomes evident that a crucial element for the estimation of dynamical inverse solutions is given by the model according to which the dynamics of the voxel currents is assumed to evolve. If a very simple model is chosen, by their model one can obtain solutions that offer only small improvements over solutions resulting from previous nondynamical (i.e., instantaneous) algorithms for solving the inverse problem; if the model contains additional information about the true dynamics, much better solutions can be obtained. Such information can at least partly be obtained by choosing a model with a sufficient flexibility for adaptation to given data; this adaptation can be performed by suitable fitting of dynamical parameters and noise covariances.

They propose to employ the maximum likelihood method for this fitting task; it becomes evident that Kalman filtering is the natural tool for calculating the likelihood

from EEG data. By assuming a somewhat more general viewpoint, they address parameter estimation as a special case of model comparison, and employ appropriate statistical criteria, namely the Akaike Information Criterion (AIC) and its Bayesian variant, ABIC, for the purpose of comparing inverse solutions. They reached numerical examples both for simulated data and for a clinical EEG recording. Finally, it should be mentioned that there exists already a sizable amount of published work dealing with applications of Kalman filtering to the analysis of EEG recordings; and furthermore there exist also applications of Kalman filtering, or related filtering approaches, to inverse problems, some of which also fall into the field of biomedical data analysis, like Electrical Impedance Tomography or evoked potential estimation (see, e.g., Baroudi et al., 1998; Kaipio et al., 1999; Karjalainen, 1997). Recently, Somersalo et al., (2003) have applied a nonlinear alternative to Kalman filtering, known as particle filtering, to the problem of estimating focal sources from MEG data. While providing important results and methodology, so far none of these studies has addressed the problem of reconstruction of distributed sources from EEG (or MEG) times series in the context of identification of optimal dynamical (i.e., predictive) models.

In their study Galka et al.,(2004), apart from the rest of the related literature, introduces a new practicable solution for the problem of applying Kalman filtering to very high dimensional filtering problems, as they arise in the case of spatiotemporal brain dynamics. Moreover, Galka et al.,(2004) replaces the largely arbitrary choice of dynamical models that can be seen in many applications of Kalman filtering, by a systematic model comparison and selection approach that provides explicit justification for the chosen model and parameters in terms of the numerical value of a statistical criterion.

By this approach, a wide class of very general models becomes available for data-driven modelling of brain dynamics.

1.1. The inverse problem of EEG generation

We start from a rectangular grid of N_v voxels covering the cortical gray matter parts of human brain; in this study, inverse solutions will be confined to these voxels. In the particular discretisation that it will be employed, there are $N_v = 3433$ cortical gray-matter voxels. At each voxel, a local three-dimensional current vector

$$j(v, t) = (j_x(v, t), j_y(v, t), j_z(v, t))^T$$

is assumed, where v is a voxel label, t denotes time, and y denotes matrix transposition. The column vector of all current vectors (i.e., for all gray-matter voxels) will be denoted by

$$J(t) = (j(1, t)^T, j(2, t)^T, \dots, j(N_v, t)^T)^T$$

which represents the dynamical state variable of the entire system.

These currents are mapped to the electroencephalographic signal (EEG), which is recorded at the scalp surface. The EEG at an individual electrode shall be denoted by $y(i, t)$, where i is an electrode label; the n_c -dimensional column vector composed of all the electric potentials at all available electrodes shall be denoted by

$$Y(t) = (y(1, t), y(2, t), \dots, y(n_c, t))^T$$

Generally in the EEG experiments, the 10–20 system is employed; all potentials refer to average reference, although other choices are possible. Due to the choice of a reference out of the set of electrodes, it is advisable to exclude one of the standard electrodes of the 10–20 system from further analysis, such that the effective dimension of Y becomes $n_c = 18$. For distributed source models, it is possible to approximate the mapping from J to Y by a linear function Baillet et al., (2001) whence it can be expressed as

$$Y(t) = KJ(t) + \varepsilon(t) \tag{1.1}$$

Here K denotes a $n_c \times 3N_v$ transfer matrix, commonly called ‘‘lead field matrix’’. This matrix can approximately be calculated for a three-shell head model and given electrode locations by the ‘‘boundary element method’’ (Ary et al., 1981; Buchner et al., 1997; Pascual-Marqui et al., 1994; Riera et al., 1998). It is an essential precondition for any approach to find inverse solutions that a reliable estimate of this matrix is available. Here we remark that typically the lead field matrix turns out to be of full rank. It will be convenient for later use to define the individual contribution of each voxel to the vector of observations by $K(v)j(v, t)$, where $K(v)$ is the $n_c \times 3$ matrix that results from extracting those three columns out of K , which are multiplied with $j(v, t)$ in the process of the multiplication of K and $J(t)$. From this definition, (1.1) can also be written as

$$Y(t) = \sum_{v=1}^{N_v} K(v)j(v, t) + \varepsilon(t) \quad (1.2)$$

By $\varepsilon(t)$, it is denoted a vector of observational noise, which is assumed to be white and Gaussian with zero mean and covariance matrix $C_\varepsilon = E(\varepsilon\varepsilon^T)$. So, the following assumption takes place that C_ε has the

simplest possible structure, namely

$$C_\varepsilon = \sigma_\varepsilon^2 I_{n_c} \quad (1.3)$$

where I_{n_c} denotes the $n_c \times n_c$ identity matrix; that is, the observation noise is uncorrelated between all pairs of electrodes and of equal variance for all electrodes (1.1) is part of the standard formulation of the inverse problem of EEG Pascual-Marqui, (1999); however in the study of Galka (2004), they interpret it as an observation equation in the framework of dynamical state-space modelling. The inverse problem of EEG generation is given by the problem of estimating the generators $J(t)$ from the observed EEG $Y(t)$; this obviously constitutes an ill-posed problem since the dimension of $J(t)$ is much larger than the dimension of $Y(t)$. As also in the case of many other

inverse problems, it is nevertheless possible to obtain approximate estimates of $J(t)$. As a representative of the numerous approaches that have been proposed for this purpose, Galka (2004) selected here the ‘‘low-resolution brain electromagnetic tomography’’ (LORETA) algorithm, proposed by Pascual-Marqui et al. (1994), as a starting point.

1.2 The Instantaneous Case

1.2.1 The LORETA approach

In this approach, a spatial smoothness constraint is imposed on discrete spatial Laplacian operator defined by

$$L = \left(I_{N_v} - \frac{1}{6} N \right) \otimes I_3 \quad (1.4)$$

Here N denotes a $N_v \times N_v$ matrix having $N_{vv'} = 1$ if v' belongs to the set of neighbours of voxel v (this set shall be denoted by $M(v)$), and 0 otherwise. By the symbol $\otimes$, Kronecker multiplication of matrices is denoted. The $(3i)$ th row vector of L acts as a discrete differentiating operator by forming differences between the nearest neighbours of the i th voxel and i th voxel itself (with respect to the first vector component). In the LORETA approach, the inverse solution is obtained by minimizing the objective function

$$E(J) = \| (Y - KJ) \|^2 + \lambda^2 \| LJ \|^2 \quad (1.5)$$

i.e., a weighted sum of the observation fitting error and of a term measuring nonsmoothness by the norm of the spatial Laplacian of the state vector. $\|\cdot\|$ denotes Euclidean norm. The hyperparameter λ expresses the balance between fitting of observations and the smoothness constraint; a nonzero value for λ provides regularisation for the solution (Hofmann, 1986). The least squares solution of the problem of minimising (1.5) is given by

$$\hat{J} = (K^T K + \lambda^2 L^T L)^{-1} K^T Y \quad (1.6)$$

here by the $\hat{J}$ estimator of the state vector J is denoted. Within the framework of Bayesian inference, this solution can be interpreted as the Maximum A Posteriori (MAP) solution for the case of Gaussian distributions for the likelihood and the prior (Tarantola, 1987; Yamashita et al., 2004):

$$\begin{aligned} p(Y|J; \sigma_\varepsilon^2) &\sim N(KJ, \sigma_\varepsilon^2) \\ p(J; \tau^2) &\sim N(0, \tau^2 (L^T L)^{-1}) \end{aligned} \quad (1.7)$$

where it is defined $\tau = \sigma_\varepsilon / \lambda$. Note that $\hat{J}$ will not depend on the reference according to which the EEG data Y was measured; this dependence is absorbed into the lead-field matrix. This effect represents another advantage of transforming EEG data into an estimated source current density: the notorious reference problem of EEG is completely removed by this transformation. The matrix $K^T K + \lambda^2 L^T L$ in (1.6) has the size $3N_v \times 3N_v = 10^4 \times 10^4$, whence actual numerical inversion is usually impracticable. The solution can be evaluated nevertheless by using the singular value decomposition of the $n_c \times 3N_v$ matrix KL^{-1} ,

$$KL^{-1} = USV^T \quad (1.8)$$

where U is an orthogonal matrix $n_c \times n_c$, S is a $n_c \times 3N_v$ matrix whose only nonzero elements are the singular values $S_{ii} = s_i$ $i = 1, \dots, n_c$, and V is an orthogonal $3N_v \times 3N_v$ matrix; only the first n_c columns of V are relevant for this decomposition, and the corresponding $3N_v \times n_c$ matrix shall be denoted by $V^{(1)}$. The matrix composed of the remaining $3N_v - n_c$ columns shall be denoted by $V^{(2)}$. After some transformations, (1.6) becomes

$$\hat{J} = L^{-1}V^{(1)} \text{diag}\left(\frac{s_i}{s_i^2 + \lambda^2}\right)U^T Y \quad (1.9)$$

Here $\text{diag}(x_i)$ denotes a diagonal matrix with elements $x_1, \dots, x_{n_c}$ on its diagonal. Numerical evaluation of this expression can be implemented very efficiently.

1.2.2 Estimation of the regularisation parameter λ

Since the inverse solution given by (1.9) will depend sensitively on the value of the hyperparameter λ , it should be chosen in an objective way. Various statistical criteria, such as Generalised Cross-Validation (GCV) Wahba (1990), and ad hoc methods, such as the L-curve approach Lawson and Hanson, (1974), have been employed for this purpose. One of the new view of point of Galka (2004) instead of these approaches, is to use the Akaike Bayesian Information Criterion (ABIC) Akaike (1980a,b), by the way one can directly compare inverse solutions obtained by different techniques by comparing their likelihood.

Given a time series of EEG observations $Y(1), \dots, Y(N_t)$, ABIC is defined as (-2) times the type II log-likelihood; that is, the loglikelihood of the hyperparameters in the context of empirical Bayesian inference. In the case of the model containing unobservable variables, the type II likelihood can be obtained by averaging the joint distribution of all variables, both observable and unobservable, over the unobservable variables, i.e., by forming the marginal distribution:

$$ABIC(\sigma_\varepsilon, \tau) = -2 \log L_{II}(\sigma_\varepsilon, \tau) = -2 \sum_{t=1}^{N_t} \log \int p(Y(t)|J(t); \sigma_\varepsilon^2) p(J(t); \tau^2) dJ(t) \quad (1.10)$$

Where $Y(t)$ are the observable and $J(t)$ the unobservable variables; σ_ε, τ are hyperparameters, and again $\tau = \sigma_\varepsilon / \lambda$. Estimators for σ_ε and λ can be obtained by maximising the likelihood given by (1.10); how this can be done in an efficient way will be presented elsewhere in more detail (Yamashita et al., 2004). Here we give only the

result of the study of Galka (2004). The type II log-likelihood $LII(\sigma_\varepsilon, \tau)$ itself can be shown to be

$$L_{II}(\sigma_\varepsilon, \lambda) = \sum_{t=1}^{N_t} \left(\sum_{i=1}^{n_c} \log \frac{s_i^2 + \lambda^2}{\lambda^2} + n_c (1 + \log 2\pi\sigma_\varepsilon^2) \right) \quad (1.11)$$

where the estimate of the observation noise variance σ_ε^2 is given by

$$\sigma_\varepsilon^2 = \frac{1}{n_c} \sum_{i=1}^{n_c} \frac{\lambda^2}{s_i^2 + \lambda^2} \bar{y}_{i(t)}^2 \quad (1.12)$$

here $\bar{y}(i, t)$ denotes the i th element of the vector $U^T Y(t)$, where U is defined in (1.8). As a result, one can obtain not only estimates for the hyperparameters, but also the possibility to calculate the ABIC value for any given inverse solution (as obtained by LORETA), i.e., an estimate for the type II likelihood. This will enable us to compare inverse solutions obtained by different techniques, since for given data the likelihood serves as a general measure of the quality of hypotheses (Akaike, 1985). It should be mentioned here that despite using an improved statistical criterion, the proper choice of the regularisation parameter remains a difficult problem of the LORETA method; in practice, frequently even the order of magnitude of the appropriate value of λ is debatable and may change drastically upon seemingly insignificant changes of the data. Also for this reason, there is a growing need for an alternative approach to estimating inverse solutions.

1.2.3 Estimation of the covariance matrix of the estimated state vector

So far, the lack of an efficient method for assessing the approximate error associated with the inverse solutions obtained by LORETA has been a serious weakness of this technique; certainly, it has contributed much to the widespread scepticism that the very idea of estimating solutions for the inverse problem of EEG generation is still facing. Strictly speaking, estimates of unobservable quantities without estimates of the corresponding error have to be regarded as meaningless. For this reason, we should

mention the method of Galka (2004) for estimating the covariance matrix C_j^{iIS} of the estimated currents $\hat{J}(t)$ of an inverse solution obtained by LORETA (here the superscript (iIS) denotes “instantaneous inverse solution”). The derivation of the expression for C_j^{iIS} is inspired by recent work by Pascal-Marqui (2002); the result is given by

$$C_j^{(iIS)} = \tau^2 L^{-1} V^{(1)} \text{diag} \left(\frac{s_i^2}{s_i^2 + \lambda^2} \right) V^{(1)T} (L^T)^{-1} \quad (1.13)$$

Through this expression, it becomes possible to display error estimates for each voxel (and each vector component) individually. Since a corresponding error estimate can also be computed for the new dynamical technique for estimating inverse solutions, another quantitative measure for the comparison of different techniques for estimating inverse solutions becomes available.

1.3 The dynamical case

1.3.1 Dynamical models of voxel currents

After discussing various aspects of the LORETA method, we shall now explain and analyze the newest method introduced by Galka (2004) proceed to the formulation of the new approach for solving the dynamical inverse problem of EEG generation; it has already been mentioned that for this purpose an additional temporal smoothness constraint could be introduced into (1.5) (Baillet and Garnero, 1997; Schmitt et al., 2002). While this approach will be pursued and extended elsewhere (Yamashita et al., 2004), here the central concept of Galka (2004) ‘s approach is a new adaptation of Kalman filtering to the case of spatiotemporal dynamics.

The Kalman filter provides the optimum tool for predicting, filtering and smoothing estimates of the state of dynamical systems that cannot be observed directly, but only through an observation equation containing observational noise (Chui and Chen, 1999).

As a presupposition for its application and as indicated in 1.part of this study, both the equations governing the dynamics and the observation equation have to be known.

Having defined a spatial discretisation by using a finite set of voxels, Galka (2004) does not formulate dynamical models also with temporal discretisation; instead, perhaps for simplicity, they regard the basic time unit of this discretisation as equal to the sampling rate of the EEG recording. The corresponding time points will be labeled by $t = 1, 2, 3, \dots, N_t$.

In general form, the dynamics of a set of N_v voxels may be described by nonlinear multivariate autoregressions given as

$$J(t) = F(J(t-1), J(t-2), \dots, J(t-p)|v) + \eta(t) \quad (1.14)$$

where p denotes the positive integer model order and $\eta(t)$ denotes dynamical noise, which we assume to be white and Gaussian with zero mean and covariance matrix $C_\eta = E(\eta\eta^T)$. When fitting such models to given data, $\eta(t)$ represents a time series of innovations, i.e., components of the data that cannot be explained from the dynamics itself. It is the aim of modelling to find dynamical models that produce a Gaussian white innovation time series, such that the process of modelling can be regarded as ‘‘temporal whitening’’. F denotes a function describing the deterministic part of the dynamics; it may depend on a vector of parameters J . This function may contain considerable internal complexity and a huge number of parameters (described by J), since it maps an input space of dimensionality $3 N_v p$ to an output space of dimensionality $3 N_v$. The simplest nontrivial example of the class of autoregressions described by (1.14) is a linear multivariate autoregressive model of first order (AR(1)):

$$J(t) = AJ(t-1) + \eta(t) \quad (1.15)$$

and here we note that the popular random-walk model is a special case of this model, with $A = I_{3N_v}$. The parameter matrix A is of size $3N_v \times 3N_v$, which in the simulations

of Galka (2004) is of the order of 10^8 . This large number of parameters is still far too high to be estimated from real data; therefore it is needed additional reductions of model complexity. Also, the practical application of Kalman filtering requires a simplified model structure. It is an arguably reasonable assumption that at short time scales, the dynamics can be restricted to local neighbourhoods; that is, each voxel will interact only with its direct spatial neighbours; in a rectangular grid of voxels, there are six direct neighbours for each voxel, except for those at the boundaries of the gray-matter parts of brain. We will discuss the physiological plausibility of Galka (2004)'s this simplification in the fourth part of our study. Hence, with their “nice” simplification most elements of A become zero.

The dynamical model for each voxel becomes

$$j(v, t) = A_1 j(v, t-1) + \frac{1}{6} B_1 \sum_{v' \in M(v)} j(v', t-1) + \eta(t) \quad (1.16)$$

where A_1 and B_1 now are the autoregressive parameter matrices (of size 3×3) for self-interaction and nearest-neighbour interaction, respectively, and $M(v)$ denotes the set of labels of gray-matter voxels that are direct neighbours of voxel v . If furthermore we assume the absence of any interactions between the three projections of local current vectors (both within and between voxels) and total spatial homogeneity and isotropy for all pairs of neighbouring voxels (this assumption extending to the three vector projections as well), ultimately the number of parameters can be reduced to two.

The dynamical model for each voxel becomes

$$j(v, t) = a_1 I_3 j(v, t-1) + \frac{1}{6} b_1 I_3 \sum_{v' \in M(v)} j(v', t-1) + \eta(t) \quad (1.17)$$

where I_3 denotes the 3×3 identity matrix; now a_1 and b_1 are scalar autoregressive parameters. This model implements complete symmetry between voxels and also between projections of local currents; but clearly, this symmetry is not preserved by

multiplication with the lead field matrix K . As a consequence of this, inverse solutions also displays nonsymmetric behaviour with respect to voxels and projections.

We capture that it is also needed additional assumptions to define the simplest possible nontrivial dynamical model, given by (1.17), and as we can easily see below that; Galka (2004), without taking into account of physiological justification, defines more mathematical assumptions to design a tractable algorithm as a starting point. By the way, it becomes possible again to consider generalisations, such as higher model order, nonlinearities, or inhomogeneities; as a consequence, it may become possible to describe more structure present in the data by the dynamical model and to relegate less power from the data to the time series of innovations $\eta(t)$. Here their aim is that the values in $\eta(t)$ be as small as possible, contain as little correlation as possible (i.e., being white noise) and have a distribution as close as possible to a Gaussian. As long as only nearest-neighbour interactions are allowed and a high degree of spatial homogeneity and isotropy is maintained, the number of unknown parameters can be kept small, and the problem remains accessible for spatiotemporal Kalman filtering.

By increasing the model order to p , it becomes possible to describe stochastic oscillations that may be present in the data; although this case represents a generalisation of (1.15) it can be incorporated into this equation by replacing the order-1 state vector $J(t)$ by $(J(t)^T, J(t-1)^T, \dots, J(t-p+1)^T)^T$, such that formally the dynamics remains a linear autoregression of first order. (1.17) can alternatively be written as

$$j(v, t) = (a_1 + b_1)I_3 j(v, t-1) + b_1 I_3 \left(\left(\frac{1}{6} \sum_{v' \in M(v)} j(v', t-1) \right) - j(v, t-1) \right) + \eta(t) \quad (1.18)$$

This equation shows more clearly that the dynamics at each voxel is composed of two contributions, the first representing the autoregressive dynamics of the voxel itself and the second representing small exogenous disturbances that partly are described as pure noise and partly as the difference between the average of the states of the neighbouring voxels and the state of the voxel itself. From (1.18) it can be seen that when compared with other spatiotemporal approaches the parameter matrix A is specially chosen as

$$A = (a_1 + b_1)I_{3N_v} - b_1 L \quad (1.19)$$

where L is the discrete spatial Laplacian operator defined in (1.4). Also when the recent related studies of brain dynamics in continuous time and space are explored, we can easily see that; different from the approach of Galka (2004), the study of Jirsa et al., (2002); and Robinson et al., (1997) directly generalise the (1.18) to partial differential equation models. In the continuous case (with respect to both time and space), the second term on the rhs of (1.18) (i.e., the term corresponding to the Laplacian) becomes a second derivative with respect to space, while the two terms $j(v, t) - (a_1 + b_1)I_3 j(v, t - 1)$ can be interpreted as a first derivative with respect to time; consequently, a standard diffusion equation results. If a model of order $p = 2$ is chosen, there will also be a second derivative w.r.t. time instead of the first derivative; in this case, a standard wave equation results. These interpretations render the model orders $p = 1$ and $p = 2$ particularly attractive.

1.3.2 Spatial whitening

As we know from the Standard Kalman Filtering Theory and as we indicated in 1. part of our study; application of Kalman filtering to the full spatiotemporal model as given by (1.15) would be infeasible in terms of computational time and memory demands due to the huge size of the parameter matrix A , i.e., if interactions between all pairs of voxels have to be considered. However, in their study Galka (2004), they have suggested to decompose this high-dimensional filtering problem into a collection of coupled low-dimensional local filtering problems, each centred at one individual voxel, as described by (1.16); only by this decomposition the spatiotemporal filtering problem becomes tractable. The low-dimensional systems remain coupled through neighbourhood interactions, but at each voxel these contributions are formally regarded as exogenous variables.

This decomposition approach is a central contribution of their new approach. To apply this decomposition to the dynamics, it is also necessary that the dynamical noise covariance matrix C_η be a diagonal matrix, as assumed in (1.3) for the case of the

observational noise covariance matrix C_ϵ . But in the case of C_η , experience obtained from the analysis of real EEG time series has shown that such assumption can typically not be justified by physiology, rather the presence of nonvanishing instantaneous correlations at least between neighbouring voxels - and therefore also between the corresponding components of the dynamical noise - has to be expected. Therefore Galka (2004) has to do an instantaneous data transformation

$$\tilde{J} = TJ \tag{1.20}$$

such that in the corresponding dynamical model

$$\tilde{J}(t) = \tilde{A}\tilde{J}(t-1) + \tilde{\eta}(t) \tag{1.21}$$

the dynamical noise covariance matrix $\tilde{C}_\eta$ becomes diagonal (here they are furthermore unplausibly assuming that all elements on the diagonal of $\tilde{C}_\eta$ are identical, a simplification that due to the large number of voxels is necessary for practical implementation; again, we capture from the related literature that this assumption is relaxed in some other works:

$$C_{\tilde{\eta}} = \sigma_{\tilde{\eta}}^2 I_{3N_v} \tag{1.22}$$

To find a simple but efficient transformation, they propose to extend the concept of temporal whitening to the spatial domain. From the Time Series Analysis theory we know that a simple whitening approach in temporal domain is given by differentiating the time series; but Galka et al. (2004) chooses to perform a spatial differentiating step (of second order) by applying the discrete Laplacian as defined by (1.4) to the dynamical state J . According to their approach, it is reasonable to assume that very fast correlations, appearing to be instantaneous with respect to the sampling time of the data, is confined to short distances, i.e., neighbouring voxels; the Laplacian represents the easiest possible choice for removing these correlations. In the fourth part of our study we will examine that, whether this is sufficiently reasonable or not. Also note that the same

transformation is employed in the 1.2.1 part (LORETA), but for a different purpose; nevertheless, this coincidence provides useful additional interpretations, as shown now. If $T = L$ is choosen, (1.21) yields

$$\tilde{J}(t) = L^{-1} \tilde{A} \tilde{J}(t-1) + L^{-1} \tilde{\eta}(t) \quad (1.23)$$

But due to (1.19) (which by definition also describes the structure of $\tilde{A}$), the matrices $\tilde{A}$ and L commute, such that by comparison with (1.15) it is seen that the choice $T = L$ corresponds to $\eta(t) = L^{-1} \tilde{\eta}(t)$, and their assumption for the nondiagonal dynamical noise covariance matrix becomes

$$C_{\eta} = L^{-1} E(\tilde{\eta} \tilde{\eta}^T) (L^{-1})^T = \sigma_{\tilde{\eta}}^2 (L^T L)^{-1} \quad (1.24)$$

By comparison with LORETA in its Bayesian interpretation (see ‘‘The LORETA method’’), it can be seen, that $\sigma_{\tilde{\eta}}$ directly corresponds to the hyperparameter $\tau = \sigma_{\epsilon} / \lambda$, such that τ^2 can be interpreted as dynamical noise variance. This quantity is meaningful also in the case of the instantaneous inverse problem and its LORETA solution; the optimal regularisation parameter becomes a measure for the ratio between the observation noise variance and the dynamical noise variance, as it should λ be. We remark that the appropriate choice of $C_{\tilde{\eta}}$ is the essential part of Galka (2004) model to fit dynamics of the model; therefore different from the related studies of dynamical inverse solutions, Galka (2004) uses statistical criterias and hence their choice of $C_{\tilde{\eta}}$ is based on improvements of a suitable statistical criterion, such as AIC. The Laplacian serves only as a first approximation, but in other researchs we see that it is also possible to define $C_{\tilde{\eta}}$ as non-stationary shock. From now on we will omit the tilde.

1.3.3 Spatiotemporal Kalman filtering

Given the observation equation, (1.1), and the dynamical equation (in the case of a linear first-order autoregression (1.21)), we could in principle apply Kalman filtering

according to its usual form; however, due to the very high dimensionality of the state variable J , this would be infeasible. But by appropriate design of certain modifications of the standard filtering procedure analyzed in 1. part of our study, it is possible to decompose the very high-dimensional filtering problem into a collection of coupled low-dimensional problems; the set of these problems is labeled by the voxel label v , i.e., it represents the spatial dimension of the problem.

Let $\hat{j}(v, t-1|t-1)$ denote the estimate of the local current vector at voxel v at time $t-1$, i.e., the local state estimate, and $p(v, t-1|t-1)$ the corresponding estimate of the local error covariance matrix (a 3×3 matrix). The notation $\hat{j}(t_1|t_2)$ (where $t_1 \geq t_2$) represents the estimate of j at time t_1 , which is based on all information having become available until (and including) time t_2 . For each voxel, the local state prediction is then given by

$$\hat{j}(v, t|t-1) = A_1 \hat{j}(v, t-1|t-1) + \frac{1}{6} B_1 \sum_{v' \in M(v)} \hat{j}(v', t-1|t-1) \quad (1.25)$$

and the corresponding local prediction error covariance matrix can be approximated by

$$P(v, t|t-1) = A_1 P(v, t-1|t-1) A_1^T + \sigma^2 \eta I_3 \quad (1.26)$$

The local state predictions $\hat{j}(v, t|t-1)$ for all voxels form the overall state prediction $\hat{j}(v, t|t-1)$ from which the observation prediction (for all electrodes) follows as

$$\hat{Y}(t|t-1) = \bar{K} \hat{J}(t|t-1) \quad (1.27)$$

The symbol $\bar{K}$ stands for the product KL^{-1} ; multiplication by the inverse of the Laplacian is needed due to the spatial whitening approach described in ‘‘Spatial whitening’’. The actual observation at time t is $Y(t)$, and the observation prediction error results as

$$\Delta Y(t) = Y(t) - \hat{Y}(t|t-1) \quad (1.28)$$

This multivariate time series represents the innovations of the actual observations, as opposed to the innovations of the (unobservable) system states, which have been denoted by $\eta(t)$ in (1.14). The corresponding observation prediction error covariance matrix can be approximated by

$$R(t|t-1) = \sum_{v=1}^{N_v} \bar{K}(v) P(v, t|t-1) \bar{K}(v)^T + \sigma_\varepsilon^2 I_{n_c} \quad (1.29)$$

Here the direct summation over voxels seems to provide the appropriate generalisation of the standard expression to the spatiotemporal case. The matrices $\bar{K}(v)$ have been defined in ‘‘The inverse problem of EEG generation’’; again the bar refers to the fact that due to spatial whitening, we have to replace K by KL^{-1} , before extracting the columns referring to voxel v . The Kalman gain matrix for voxel v follows as

$$g(v, t) = P(v, t|t-1) \bar{K}(v)^T R(t|t-1)^{-1} \quad (1.30)$$

and finally the local state estimation and the corresponding local estimation error covariance matrix are given by

$$\hat{j}(v, t|t) = \hat{j}(v, t|t-1) + g(v, t) \Delta Y(t) \quad (1.31)$$

and

$$P(v, t|t) = (I_3 - g(v, t) \bar{K}(v)) P(v, t|t-1) \quad (1.32)$$

respectively. This set of equations constitutes the new spatiotemporal Kalman filter. It should be stressed that Eqs. (1.25), (1.26), (1.30), (1.31) and (1.32) are applied locally to each voxel, whereas only (1.27) requires a large-scale multiplication of the lead-field matrix $\bar{K}$ with the full $(3N_v)$ -dimensional state vector $\hat{J}$. For practical application of this filter to time series data, initial values $\hat{j}(v, 1|1)$ and $p(v, 1|1)$ are needed. As initial state

estimates $j(v,1|1)$, whether it is chosen psycholological theory based - guessed value or, as Galka (2004) does, is chosen the solutions provided by the LORETA algorithm, as presented in 1.2.1 part ‘‘The LORETA approach’’; the hyperparameters are chosen by minimisation of ABIC. Since it is impracticable to perform this optimisation directly in the $(3 N_v)$ -dimensional state space, we apply the optimisation to the $(n_c p)$ -dimensional observation space of the first p observations $Y(1), \dots, Y(p)$ and use the LORETA technique for mapping points in the observation space to new prospective initial states. As we can easily see, concerning the choice and optimisation of initial values, the study of Galka (2004) implements Kalman Filter differently from more conventional applications of Kalman filtering, which usually do not employ this extended estimation approach. However, their extended estimation approach is essentially based on certain assumptions which are neither psychologically plausible nor statistically sufficient.

1.3.4 Estimation of the covariance matrix of the estimated state vector

(1.32) provides us with covariance estimates for the reconstructed states (given by (1.31)); we may use these estimates as a measure for the error of each component of the estimated state vectors. Due to the spatial whitening transformation (see 1.3.2 ‘‘Spatial whitening’’) the covariance matrix of the actual currents is given by

$$C^{(dIS)}_{L^{-1}\hat{J}} = L^{-1} P(t|t) (L^T)^{-1} \quad (1.33)$$

where the superscript (dIS) denotes ‘‘dynamical inverse solution’’. By $P(t|t)$ the complete covariance matrix of the estimated states is denoted, i.e., a $3N_v \times 3N_v$ matrix the diagonal of which consists of the $P(t|t)$ matrices given by (1.32) for each voxel. The diagonal elements of $C^{(dIS)}_{L^{-1}\hat{J}}$ provide time-dependent variances for each element of the complete estimated state vector $L^{-1}\hat{J}$ (again the premultiplication with L^{-1} is necessary due to spatial whitening).

1.3.5 Silent and observable sources

In the (instantaneous) inverse problem of EEG generation in general researchers are facing the apparently hopeless task to estimate a set of more than 104 unknown quantities from usually less than 102 observations. As a condition for identifying a unique solution, additional constraints have to be defined and imposed, such as the smoothness constraint of LORETA. Such constraints may succeed to render the inverse problem well-posed, but at the cost of a lack of justification, e.g., in terms of physiology. The inherent predicament of inverse problems, i.e., the very unfavourable ratio between the numbers of known and of unknown quantities, is avoided, but not solved by such constraints. The central question remains unsolved: How can we expect that all relevant information about the spatially distributed primary current density could be reconstructed from a small number of surface measurements? In this section, we will try to regard the answers cited in the related “dynamical inverse problem” literature by comparing them. The problem can be expressed in quantitative form by considering the observation equation (1.1) and the singular value decomposition of the lead field matrix (1.8):

$$Y(t) = \bar{K}J(t) + \varepsilon(t) = USV^T J(t) + \varepsilon(t) = USH(t) + \varepsilon(t) \quad (1.34)$$

Here it is defined $H(t) = V^T J(t)$, i.e., Baroudi et al., (1998) ; Ozaki et al., (2000) and Galka et al.,(2004) have applied an orthogonal transform to the state vector $J(t)$. As mentioned already in the 1.2.1 part : “The LORETA approach”, the $n_c \times 3N_v$ matrix S is composed of a $n_c \times n_c$ diagonal matrix containing the singular values (which are all nonzero since the lead field matrix has full rank), while the remaining $(3N_v - n_c)$ columns contain only zeros. Therefore only the subspace spanned by the first n_c elements of the transformed state vector $H(t)$ is mapped to the observation vector $Y(t)$, while the subspace of the remaining elements is completely ignored. Components of the “true” state $J(t)$, which by the orthogonal transformation are mapped into the former subspace, represent “*observable sources*”, whereas those components that are mapped

completely into this latter subspace cannot be observed, whence they are termed “*silent sources*” (note that such “sources” are not localised in physical space). Within the framework of the instantaneous inverse problem, there exists no way to obtain information about these state components. Here we would like to argue that the situation is much different in the case of the dynamical inverse problem. However counterintuitively, Galka et al.,(2004) claims that: “*if the problem of estimating unobserved quantities is treated in a state space framework, it can be easily shown that under certain circumstances information from the subspace of silent sources will propagate into the subspace of observable sources*”. To demonstrate this effect, these authors apply the orthogonal transformation $H = V^T J(t)$ to (1.15) and obtain

$$H(t) = V^T A V H(t-1) + V^T \eta(t) = \tilde{A} H(t-1) + \tilde{\eta}(t) \quad (1.35)$$

where $\tilde{A}$ and $\tilde{\eta}_t$ denote the transformed transition matrix and the transformed dynamical noise vector, respectively. Let $(H^{(1)}(t)^T, H^{(2)}(t)^T)^T$, such that $H^{(1)}(t)$ denotes the first n_c elements of the vector $H(t)$ and $H^{(2)}(t)$ the remaining elements. Then, $H^{(1)}(t)$ represents the subspace that is mapped to the observations, i.e., the subspace of observable sources, while $H^{(2)}(t)$ represents the subspace of silent sources. The same subdivision is also applied to the transformed transition matrix $\tilde{A}$:

$$\begin{pmatrix} H^{(1)}(t) \\ H^{(2)}(t) \end{pmatrix} = \begin{pmatrix} \tilde{A}^{(1,1)} & \tilde{A}^{(1,2)} \\ \tilde{A}^{(2,1)} & \tilde{A}^{(2,2)} \end{pmatrix} \begin{pmatrix} H^{(1)}(t-1) \\ H^{(2)}(t-1) \end{pmatrix} + \tilde{\eta}(t) \quad (1.36)$$

such that $\tilde{A}^{(1,1)}$ and $\tilde{A}^{(2,2)}$ are $n_c \times n_c$ and $(3N_v - n_c) \times (3N_v - n_c)$ matrices, respectively.

From this equation, it can be seen that the “partial” transition matrix $\tilde{A}^{(1,2)}$ plays a crucial role, since it maps the subspace of silent sources to the subspace of observable sources. It is through this pathway that information from the silent sources is propagated into the observable sources, and from there into the observations. Obviously, this propagation of information will not take place if $\tilde{A}^{(1,2)} = 0$. On the contrary, to have

information from all elements of $H^{(2)}(t)$ be propagated into $H^{(1)}(t)$ each column of $\tilde{A}^{(1,2)}$ must contain at least one nonzero element. From the analyses of Galka (2004) we can easily derive the core idea that, these authors assume that, the *current* sources behave as if they are stationary AutoRegressive sources. Even we can not find this assumption physiologically plausible, as a fundamental idea of rest of our study we can consider the special case of $A = I_{3N_v}$, i.e., a random-walk type dynamics without neighbour interactions. Then, $\tilde{A} = V^T A V = V^T V = I_{3N_v}$ and consequently $\tilde{A}^{(1,2)} = 0$.

If, on the other hand, we choose a transition matrix with nondiagonal elements, such as given by (1.19), $V^T A V$ will not be diagonal and $\tilde{A}^{(1,2)} \neq 0$. Using the partition of V into $V^{(1)}$ and $V^{(2)}$, as defined in ‘‘The LORETA approach’’, it can be seen that

$$\tilde{A}^{(1,2)} = \left(V^{(1)T} A \right) V^{(2)} \quad (1.37)$$

Due to the orthogonality of V , we have $V^{(1)T} V^{(2)} = 0$, i.e., the columns of $V^{(1)}$ are orthogonal to the columns of $V^{(2)}$; but multiplication by a nondiagonal matrix A will replace the columns of $V^{(1)}$ by a set of n_c different columns that generically are no longer orthogonal to any of the columns of $V^{(2)}$. Therefore we easily capture that, in general related ‘‘dynamical inverse solution’’ studies presume that generically all elements of $\tilde{A}^{(1,2)}$ will be nonzero. This argument is important for our study because, in the following parts we will show why these studies intuitively make the same mistake. Consequently, one can expect that there will be a flow of information from all elements of $H^{(2)}(t)$ into $H^{(1)}(t)$. This argument shows that only by using a dynamical model including nonvanishing neighbour interactions, state components belonging to the subspace of silent sources become accessible for reconstruction by the spatiotemporal Kalman filter.

1.3.6 Observability in state space models of brain dynamics

In the previous section, we examined certain approaches and outlined the reasons why the spatiotemporal Kalman filtering approach is found capable of accessing information about unobservable state components in the literature; now we would like to mention that there exists a rigorous theory addressing the question of whether for a given model of the dynamics and the observation the unobserved quantities can be reconstructed. This theory is built around the central concepts of observability and controllability (Kailath, 1980; Kalman et al., 1969). Assume that we are dealing with a dynamical system that evolves according to linear dynamics as described by (1.15) (with a constant transition matrix A), and that we are observing this system through an observation equation like (1.1) (with a constant observation matrix $\bar{K}$). If it is possible to reconstruct the true states of the system from the observations, the pair $(A, \bar{K})$ is said to be “*observable*”.

Various tests for observability of dynamical systems have been suggested in the literature. A well-known test states that the pair $(A, \bar{K})$ is observable, if and only if the observability matrix O , being defined by

$$O = \begin{bmatrix} \bar{K}^T & A^T \bar{K}^T & (A^T)^2 \bar{K}^T & \dots & (A^T)^{3N_v-1} \bar{K}^T \end{bmatrix}^T \quad (1.38)$$

has full rank, $\text{rank}(O) = 3N_v$ (Kailath, 1980). Here N_v denotes the dimension of the state vector, i.e., the number of unknown quantities. In the case of the dynamical inverse problem of EEG generation, this matrix has the size $3N_v n_c \times 3N_v = 185,382 \times 10,299$ (when using the corresponding values of the spatial discretisation as employed in the study Galka (2004)), which is by far too large for numerical calculation of the rank. Kalman filtering and observability theory are usually not applied to problems of this size. For this reason, we are currently not yet able to present a rigorous proof of observability for their algorithm. On the other hand, observability constitutes the essential precondition for the reconstruction of the unobserved states by Kalman filtering, i.e., for the identification of a unique inverse solution. Interestingly without

rigorously proving observability Galka (2004) demonstrates by application to simulated and to real EEG data that Kalman filtering can be applied successfully to the dynamical inverse problem of EEG generation. This means that these authors again assumptionally presume observability of the pair $(A, \bar{K})$ is given. We find this approach again counterintuitive and rather abstract and we critique this approach in the following part of our study.

1.3.7 Parameter estimation

If we choose the model as autoregressive like done generally in the literature, then the general autoregressive model described by (1.14) depends on a parameter vector v ; in the largely simplified model given by (1.17), we have $v = (a_1, b_1)$, and autoregressive models of higher order have $v = (a_1, \dots, a_p, b_1, \dots, b_q)$; here we are permitting the possibility of choosing $p \neq q$ i.e., choosing different autoregressive model orders for self-interaction and nearest-neighbour interaction. Usually, there will be no detailed prior knowledge about appropriate values for these parameters available. Furthermore, we do need estimates for the variances σ^2_ϵ and σ^2_η , as defined by Eqs. (1.3) and (1.24). Estimates for these dynamical parameters and variances should be obtained preferably from actual data. This can be accomplished within the framework of spatiotemporal Kalman filtering by likelihood maximisation. So far, to the best of our knowledge, only Galka (2004) made a successful application of the principle of likelihood maximisation to the field of inverse problems; but, recently, we can see that Phillips et al. (2002) have presented an approach involving restricted maximum likelihood, but their approach does not involve dynamical modelling. So we can cite this newest application of Galka (2004) as follows: Assume that an EEG time series $Y(t)$ is given, where $t = 1, 2, \dots, N_t$. At each time point, the Kalman filter provides an observation prediction $\hat{Y}(t)$, given by (1.27), and hence also observation innovations $\Delta Y(t)$; if for these a multivariate Gaussian distribution with mean $\hat{Y}(t)$ and covariance matrix $R(t|t-1)$ is assumed, the logarithm of the likelihood (i.e., log-likelihood) immediately results as

$$\log L(v, \sigma_\varepsilon^2, \sigma_\eta^2) = -\frac{1}{2} \sum_{t=1}^{N_t} ((\log |R(t|t-1)|) + \Delta Y(t)^T R(t|t-1)^{-1} \Delta Y(t) + n_c \log(2\pi)) \quad (1.39)$$

Here $| \cdot |$ denotes matrix determinant. The log-likelihood is known to be a biased estimator of the expectation of Boltzmann entropy (Akaike, 1985); only a small further step is needed for the calculation of an improved unbiased estimator of (-2) times Boltzmann entropy, the well-known Akaike Information Criterion (AIC) (Akaike, 1974):

$$AIC(v, \sigma_\varepsilon^2, \sigma_\eta^2) = -2 \log L(v, \sigma_\varepsilon^2, \sigma_\eta^2) + 2(\dim(v) + 2) \quad (1.40)$$

where $\dim(v)$ denotes the number of parameters contained in the parameter vector v ; it is further increased by 2 due to the need to fit σ_ε^2 and σ_η^2 from the data.

1.4 GARCH modelling of covariance in dynamical estimation of inverse solutions

In this part of our study, we examine a related issue for modelling dynamical inverse problems. As we indicated in the second part of our study, very new approaches criticising the instantaneous inverse solutions, claim that spatiotemporal kalman filtering is a very efficient way to derive the more realistic solutions to dynamical inverse problems. However, contrast to these “optimistic ideas”, as we see, these new approaches concerns very unplausible assumptions within physiological datas. Behind this, there is another aspect that all the mentioned methods, both instantaneous and spatiotemporal methods, lack to model dynamical EEG inverse problems: This is the absence of *non-stationarity* in EEG data. Favorably, after the study of Galka (2004), certain authors found it very necessary to capture the non-stationarity of the EEG data within spatiotemporal solutions.

We will examine and analyze here the most important of these studies; as because in our original study cited in fourth part, we use non-stationary model to explain the behaviour of EEG data, we consider that it is useful to regard related literature briefly which extended the spatiotemporal solutions to “dynamical inverse problems” by adding non-stationarity in observed data. We think that, this brief analysis can shed light on the

importance of our original analysis cited in the fourth part. Firstly, we briefly explain the idea and necessity of adding non-stationarity to the EEG data which is “assumed” to be autoregressive in spatiotemporal solutions as we analyzed in second part of our study, and their contributed solutions to the literature.

Very contemporarily, the method for capturing the non-stationarity in observed EEG data within spatiotemporal solutions is ARCH/GARCH method. As we indicated in second part of our study while examining the existing literature, that there are very strong assumptions such as time-invariance of error or innovation covariance and as homogeneity and as neighbourhood constraints for deriving unobserved states of the system with spatiotemporal kalman filtering (newest method in related research area). Even if these strong assumptions are necessary to model the unobserved system mathematically, physiological data show us that, these assumptions are not very plausible and intuitive. Hence, certain researchers tried to relax some of these assumptions. As it is related to our original study, we will here regard one of these new extensions of spatiotemporal solutions which replaces the time – invariance of error covariance with time – varying error covariance and by the help of this replacement that succeed to add “non-stationarity” in EEG data. In fact, this approach is more realistic. In part 1.4.1 of our study we will first briefly explain the mathematical foundations of this time-varying error covariance approach, this is called “*ARCH/GARCH processes*”, and then we analyse and criticise its adaptation to spatiotemporal solutions of dynamical EEG inverse problems.

We From Econometrics, since 1982 we know that this ARCH/GARCH processes have been successfully used to model non-stationarity in covariance of errors or innovations or shocks especially in Macroeconomics and Financial Data Analysis research areas. This elegant approach “*generalised autoregressive conditional heteroskedasticity*” (*GARCH*) model provides more freedom to the covariance matrix in the context of state estimation, and originally introduced in Econometrics as a model for time-varying volatility (and recently honoured with the Nobel Prize in Economic Sciences 2003).

The idea of applying a Kalman filter to GARCH models was proposed by Harvey et al.; here in part 1.4.2 we will analyze a different point of view which is introduced by Yamashita et al.(2004) by regarding the combination of a Kalman filter with a covariance model of GARCH type as a new variant (in fact, generalisation) of Kalman filtering. We find useful to examine this nice approach because this new generalised Kalman filter can be applied to a wide variety of dynamical models and data sets. Since covariance matrices represent a core element of the Kalman filter iteration, the incorporation of the GARCH model into the filter constitutes a major modification of the standard Kalman filter. For numerical results and simulation examples of inverse problem of EEG generation one can refer to (Yamashita et al. 2004).

After we finished the very brief description of ARCH and its generalised version GARCH processes in a very general framework and after we gave certain fundamental theorems of this processes introduced by famous economist R.Engle; we can now analyze why these types of processes needed to be introduced in EEG modelling researches and Brain Mapping areas, and also examine how these types of processes are used for “more realistic” estimation of unobserved sources in brain within a theoretical context following from very contemporary literature. This examination is essential for our original study cited in the fourth part because, as one can easily see, our results do not match with the results claimed in these new literature of related research areas.

1.4.1 Dynamical Estimation of Inverse Solutions of EEG Time Series by GARCH

Method

In part (1.2.3) the spatiotemporal Kalman filter has been developed within the context of the inverse problem of EEG generation; here we will summarise the main steps in generalised notation. Then we will introduce GARCH process in the model explained in part 1 of this study and moreover following from Yamashita et al. (2004) we will examine the solution of this dynamical inverse EEG problem in the framework of a “more strong” apriori assumption on the nature of unobserved sources. This examination is essential for our original study because, in the related research literature we know that, the newest studies use this “strong assumption”; however, in the fourth part of this study

we show that, the assumption that certain researchers made about the nature of unobserved sources located in the gray matter of brain is strictly false .

In next section, we examine that how the time – variant noise or error or shock or innovation could be introduced in such a spatiotemporal model, and in this context how the generalised kalman filter can be used to estimate the locations of unobserved current sources in gray – matter of brain. Nowadays, even if this method is found very new and succesful for brain mapping, in the fourth part of this study we will show that both intuitively and physiologically these reuslts are no longer valid.

1.4.1.1 GARCH modelling of covariance¹

Application of the Kalman filter, as described in the previous section, anticipates that estimates of the parameters $a_1, a_2, B, \sigma_\epsilon^2$, and σ_η^2 are available; in part (1.2.7) it has been described, how such estimates can be obtained from given data by likelihood maximisation.

This is feasible due to the implicit homogeneity and stationarity assumptions, according to which the values for these parameters do not depend on v and k , i.e., on spatial location and time. If for certain parameters these assumptions are relaxed or dropped, improved inverse solutions can be obtained, but the parameter estimation step becomes considerably more difficult, since the number of parameters to be estimated will assume much larger values.

As an alternative we suggest to include the dynamical noise variance σ_η^2 into the local state, thereby making it dependent on space and time:

$$\mu(v, k) = (j(v, k), j(v, k - 1), \sigma_\eta^2(v, k)) \quad (1.41)$$

it has been known for long that an appropriate choice of σ_η^2 is particularly important in Kalman filtering, whereas the other parameters are less critical. The dynamics of

¹ For a nice application of this method to EEG data refer to **Wong and Ozaki et al.(2005)**,

$\sigma_\eta^2(v, k)$ could be modelled again by a stochastic autoregression, as it is sometimes done in “stochastic volatility models” in econometrics financial research area; however, this would render the filtering process considerably more difficult. The central idea of GARCH modelling is to employ the prediction error obtained at the *previous* time step as an estimate of the stochastic shocks driving the dynamics of the time-dependent covariance. Since for inverse problems these prediction errors (in state space, i.e., at the local grid points) are not directly available, we suggest to propagate the information contained in the observation prediction errors back into state space by using the corresponding Kalman gain, i.e., using the previous value of the local state prediction error $g(v, k - 1)\Delta \mathbf{Y}(k - 1)$ instead; this approach represents the consistent application of the basic idea of GARCH modelling to the state-space case. Among numerous possible choices for the GARCH dynamics, we choose a variant based on logarithms introduced by D.B. Nelson (1991), given by

$$\log \sigma_\eta^2(v, k) = \log \sigma_c^2 + \alpha \log \sigma_\eta^2(v, k - 1) + \gamma \log \sum_{i=1}^{n_x} [g(v, k - 1)\Delta Y(k - 1)]_i^2 \quad (1.42)$$

the summation over (squared) vector components (labelled by i) represents the simplest possibility to adapt a vector-valued state dynamics to a scalar covariance dynamics.

2 MODELLING CLINICAL EPILEPSY EEG WITHOUT ANY APRIORI ASSUMPTION: INTEGRATED PROCESS

This part of our study concerns an original investigation of clinical EEG recordings. The data are taken from the individuals who are under the suspect of having Alzheimer and from the individuals who are thought to be subject to Epilepsy. The EEG of persons with epilepsy can be divided into two categories, the *interictal* and *ictal* EEG. The interictal (routine) EEG is the EEG recording taken when the patient is not having seizures. Most patients with seizures will have at least one routine EEG. This test is done to look for interictal epileptiform abnormalities, that is, abnormal activity that can occur in a patient with epilepsy in the absence of an actual seizure. Sometimes the patient is asked to attempt to go to sleep, or to hyperventilate, or a strobe light is flashed in his or her eyes to try to bring out these abnormalities. Finding these abnormalities confirms that the patient has seizures, and helps the doctor determine what type they are.

To the best of our knowledge, both in the Epilepsy research literature and Alzheimer research literature, *there exists no study and no analysis which explores Brain EEG recordings in the context of time – series analysis* without any apriori assumption. With the phrase “ without any apriori assumption” we claim that: As we can easily see from the similiar studies compiled in the second and third parts of this thesis, researchers make some apriori “strong assumptions” over the nature of unobserved sources; however, here we want to examine the Brain signals whether they are non-stationary or stationary in their nature and by the way we plan to reach some arguments after this “objective” analysis.

Up to our knowledge there may be several reasons for researchers not to perform time series analysis to the Brain EEG recordings. These reasons can be as follows:

1. Technically, after the discovery of cardiac signal, researchers seek for some pattern signals in the Brain EEG, which are assumed to shed light on non-variant relations between the events occurring in human brain.
2. Since the foundations of Biomedical research have been based on engineering methods and the goal of these researchs is mainly to obtain the map of signal sources in human brain for the usage of medical specialists; in general the related research have been constrained with certain signal processing techniques.

Our point of view and approach are completely different. We analyze EEG recordings in time domain and we apply certain statistical tests to them to better understand their nature. Our examination method to do these analysis can be summarized as follows: Firstly, we explore the stationarity of clinical EEG data obtained from certain individuals using some statistical and contemporary methods. As the number of samples recorded by EEG is huge for statistical methods, we also analyze the stationarity of these signals in small sample intervals, that means we separate our analysis in two parts:

Short – Run and Long – Run analysis. Secondly, if we find that some signals are non – stationary in short -run, then we plan to investigate the behaviour of these signals in the long-run. That means, we search whether there is a relation between the unobserved signal sources which generate non-stationary data or not. If there exists a statistical relation between these sources then what is its nature and how can we comment this relation medically? Thirdly, if there exists a long-run or short – run relationship between unobserved signal sources, then we seek whether this relationship is stationary or not; that means whether there is *Co-Integration* between the signals generated by unobserved signal sources. Finally, if we discover such a relationship then we extend our anlaysis to explore the possibility to estimate future values of these brain signals. The latter possibility is worthwhile to explore because, this can give us the “key” to better estimate the location of “**diseased – sources**” in the gray matter of Brain and hence to estimate the Epilepsy Crises and Spikes.

To answer all of these questions, we first briefly explain theoretical foundations of Non-stationary time series analysis, and its various statistical test methods that one of them

also used by us in this thesis. The subject ordering is roughly as follows; Non – stationary data analysis, Integrated processes, test of non – stationarity and test methods that we use for this study.

We also include this study the notion *long-run equilibrium* which is commonly used in econometrics because, if we find non-stationarity in EEG recordings then we have to analyze the long-run behaviours of their generators and the relationships between them; in econometrics literature this long-run behavior case is always examined under the notion “Equilibrium”. Because it is a must to obtain equilibrium(s) to make statistical estimations. The notation that we use here can be viewed a little bit “relaxed” , but it is the one widely accepted in macroeconomics, financial data analysis and econometrics - statistics literature.

2.1 Non-Stationary Data Analysis

A process x_t is stationary² if and only if :

1. $E(x_t) = m$ for $\forall t$ $m \in R$ constant
2. $Var(x_t) = \sigma^2$ for $\forall t$ σ^2 is constant
3. $Cov(x_t, x_{t-h}) = \gamma(h)$ $\forall t, h$ certain function of $\gamma(h)$

in which $E(\bullet)$ is *Expected Value Operator*.

If one of these properties can not be reached, one can say that the stationarity of the process x_t is violated.

After we recall the definition of weak – stationarity, we begin to analyze the types and the characteristics of Non-Stationarity.

2.1.1 Types of Non-Stationarity

First classification of Non-Stationarity can be established according to a differentiation between Deterministic Non-Stationarity and Stochastic Non-Stationarity.

²Hayashi, F. 2000, *Econometrics*, Princeton University Press

2.1.1.1 Deterministic Trend Stationary

Definition: A process x_t , $t \in Z$, such that

$$x_t = \alpha + \beta.t + \Psi(L)\varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma^2)$$

where $\Psi(L)\varepsilon_t$ figures out a process linear stationary is called a Deterministic Trend Stationary Process (TS). Here L is defined as *Lag Operator*.

This approach corresponds to the classical additive decomposition of a process at polynomial tendance and stationary process.

This process violates the first criteria of Stationary Conditions cited above. So, when we substract time trend $(\beta.t)$ from the process, the process (x_t) becomes Stationary.

2.1.1.2 Difference Stationary

Definition : A process x_t , $t \in Z$, such that

$$(1 - L)x_t = \alpha + \Psi(L)\varepsilon_t$$

where $\Psi(L)\varepsilon_t$ figures out a process linear stationary is called a Difference Stationary Process (DS). Here L is defined as *Lag Operator*.

This process violates the second and the third criteria of Stationarity Conditions cited above. By definition, when we substract process x_{t-1} from the process x_t , we obtain a linear Stationary Process. So we call these type of processes as Difference Stationary Process.

We can give a simple example for this type of process, which is more classical and well-known in Statistics.

Random Walk:

Definition: Let $(\varepsilon_t)_{t \in N}$ be a White Noise, $\varepsilon_t \sim N(0, \sigma^2)$. The sign “Tilda” signifies the distribution. The process $(x_t)_{t \in N}$

$$x_t = \alpha + \sum_{i=0}^s \varepsilon_t$$

is called Random Walk with Drift (α).

2.1.2 Another approach for comparing these two type process:

In order to better comparison of the two formes of Non-Stationarity, let's consider a linear process with a deterministic component.

$$x_t = \alpha + \beta.t + \left[\frac{\Theta_q(L)}{\Phi_p(L)} \right] \varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma^2)$$

Proposition 2.1: If the polynomial $\Phi_p(z)$ transformed from the process $\Phi_p(L)$ has all of its roots out of the unit-circle with raidus ($z=1$), then the stochastic part of the process x_t is stationary and the process x_t can be written under the form Trend Stationary (TS)

$$x_t = \alpha + \beta.t + C(L)\varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma^2)$$

and where $C(L) = \Phi_p^{-1}(L) \cdot \Theta_q(L)$

However, if the polynomial $\Phi_p(z)$ has one root that equals to 1, then let $\Phi_p(L)$ be

$$\Phi_p(L) = (1-L)(1 - \hat{\phi}_1 L - \hat{\phi}_2 L^2 - \dots - \hat{\phi}_{p-1} L^{p-1}) = (1-L)\hat{\Phi}_{p-1}(L)$$

and hence, the process x_t can be written under the form of Difference Stationary (DS)

$$(1-L)x_t = \Delta x_t = x_t - x_{t-1} = \alpha + \hat{C}_\infty(L)\varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma^2)$$

and where $C_\infty(L) = \hat{\Phi}_{p-1}^{-1}(L) \cdot \Theta_q(L)$

2.1.3 Consequences of TS and DS representations

2.1.3.1. The Prevision

Proposition 2.2³: Let $(x_t)_{t \in \mathbb{Z}}$ be a process with nature of Trend Stationary (TS)

$$x_t = \alpha + \beta.t + C(L)\varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma^2)$$

Then s - period ahead prevision and s - period ahead prevision error of the process x_t can be written respectively as:

$$\hat{x}_t(s) = \alpha + \beta(t + s) + c_s \varepsilon_t + c_{s+1} \varepsilon_{t-1} + \dots \text{where}$$

$$C(L) = c_0 + c_1.L + c_2.L^2 + \dots + c_s.L^s$$

$$E(x_{t+s} - \hat{x}_t(s))^2 = (1 + c_1^2 + c_2^2 + c_3^2 + \dots + c_{s-1}^2) \sigma^2$$

Two consequences:

1. When the prevision horizon ($s \rightarrow \infty$) tends to infinite, the TS process tends to an quadratic average to the deterministic tendance $(\alpha + \beta(t + s))$.
2. Also, the prevision error converges to a finite limite.

Proposition 2.3: Let $(x_t)_{t \in \mathbb{Z}}$ be a process with nature of Difference Stationary (DS)

$$(1 - L)x_t = \alpha + C(L)\varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma^2)$$

Then s - period ahead prevision and s - period ahead prevision error of the process x_t can be written respectively as:

$$\hat{x}_t(s) = s.\alpha + x_t + (c_s + c_{s-1} + c_{s-2} + \dots + c_1)\varepsilon_t + (c_{s+1} + c_s + \dots + c_2)\varepsilon_{t-1} + \dots$$

$$E(x_{t+s} - \hat{x}_t(s))^2 = (1 + (1 + c_1)^2 + (1 + c_1 + c_2)^2 + (1 + c_1 + c_2 + c_3)^2 + \dots + (1 + c_1 + c_2 + \dots + c_{s-1})^2) \sigma^2$$

³ Banerjee et al.,1993"Co-Integration, Error Correction, and The Econometric Analysis of Non-Stationary Data" Oxford University Press,

Two results:

1. When the prevision horizon $(s \rightarrow \infty)$ tends to infinite, the DS process converges to itself and to a line which modifies itself.
2. Also, the prevision error does not converge.

2.1.3.2 The impact of long-term multipliers:

We know that the multiplier of order $-s$ is defined for a linear process by $\frac{\partial x_{t+s}}{\partial \varepsilon_t}$

Proposition 2.4: The impact of the multiplier of order $-s$ will be

$$x_t: \quad TS \rightarrow \frac{\partial x_{t+s}}{\partial \varepsilon_t} = c_s \xrightarrow{s \rightarrow \infty} 0$$

$$x_t: \quad DS \rightarrow \frac{\partial x_{t+s}}{\partial \varepsilon_t} = 1 + c_s + c_{s-1} + \dots + c_1 \xrightarrow{s \rightarrow \infty} C(1)$$

where $C(1)$ is the value of first lag; that is $L = 1$ in $C(L)$.

Although, in the long-term, within the TS process, the impact of a shock or innovation at any instant t disappears; within the DS process a shock or innovation at any instant has a permanent effect.

2.1.4 Brownian Motion

In this part, we will mention Brownian motion and briefly give its theoretical foundations, that is the core theoretical idea behind Non-Stationary data analysis and examine the properties of its special form: *Random Walk*. We will explain the fundamental notions briefly, and as because it is originally concerned with the estimation of Non-Stationary data, we will not profoundly study it.

Consider a random walk,

$$x_t = x_{t-1} + \varepsilon_t \tag{2.1}$$

in which errors or shocks or innovations are standard Normal variables i.i.d distributed and assume that:

$$\varepsilon_t \sim N(0,1)$$

If the process x_t is started with $x_0 = 0$, then it follows that

$$x_t = \varepsilon_1 + \varepsilon_2 + \varepsilon_3 + \dots + \varepsilon_t \quad \text{which means} \quad x_t \sim N(0,t)$$

Moreover, the change in the value of x between instants t and s ,

$$x_s - x_t = \varepsilon_{t+1} + \varepsilon_{t+2} + \dots + \varepsilon_s$$

is itself distributed $N(0, (s-t))$ and is independent of the change between instants r and q for any dates $t < s < r < q$.

Consider the change between x_{t-1} and x_t . This innovation ε_t was taken to be $N(0,1)$.

Suppose we view ε_t as the sum of two independent Gaussian variables:

$$\varepsilon_t = e_{1t} + e_{2t} \quad \text{where} \quad e_{it} \sim N\left(0, \frac{1}{2}\right).$$

We might then associate e_{1t} with the change between x_{t-1} and the value of x_t at the some interim point say $x_{t-\frac{1}{2}}$; that is,

$$x_{t-\frac{1}{2}} - x_{t-1} = e_{1t} \tag{2.2}$$

and e_{2t} with the change between $x_{t-\frac{1}{2}}$ and x_t ;

$$x_t - x_{t-\frac{1}{2}} = e_{2t} \tag{2.3}$$

Sampled at integer instants $t = 1, 2, 3, \dots$, the process of (2.2) and (2.3) will have exactly same properties as (2.1), since

$$x_t - x_{t-1} = e_{1t} + e_{2t} = \varepsilon_t \sim N(0,1)$$

In addition, the process of (2.2) and (2.3) is defined also at the non-integer dates

$\left\{t + \frac{1}{2}\right\}_{t=0}^{\infty}$ and retains the property for both integer and non-integer dates that

$(x_s - x_t) \sim N(0, s - t)$ with $(x_s - x_t)$ independent of the change over any other nonoverlapping interval.

By the same reasoning, we could imagine partitioning the change between $(t-1)$ and t into N separate sub-periods:

$$x_t - x_{t-1} = e_{1t} + e_{2t} + \dots + e_{Nt} \quad \text{where} \quad e_{it} \sim N\left(0, \frac{1}{N}\right)$$

The result would be a process with all the same properties as equation (2.1), defined at finer and finer grid of dates as we increase N . The limit as $N \rightarrow \infty$ is a *continous-time* process known as *Standard Brownian Motion*, also called as *Wiener Process* and the value of this process at date t is denoted by $W(t)$. A continous-time process is a random variable that takes on a value for any non-negative real number t , as distinct from a discrete-time process, which is only defined at integer values of t . To emphasize the distinction from now on, we will put the date in parantheses when describing the value of a continous-time variable at date t (as in $W(t)$) and use subscriptps as in before for a discrete-time variable (as in x_t). A discrete-time process was represented as a countable sequence of random variables, denoted $\{x_t\}_{t=1}^{\infty}$. A realization of a continous-time process can be viewed as a stochastic function, denoted $W(\bullet)$,

where $W : t \in [0, \infty) \rightarrow R^1$.

A particular realization of Brownian motion turns out to be a continous function of t , because the change within the process between t and $t + \Delta t$ is distributed $N(0, \Delta)$. Such a change is essentially certain to be arbitrarily small as the interval Δ tends to 0.

*Definition*⁴: Standard Brownian Motion $W(\bullet)$ is a continuous-time stochastic process, associating each date $t \in [0,1]$ with the scalar $W(t)$ such that:

- a) $W(0) = 0$
- b) For any dates $0 \leq t_1 < t_2 < t_3 < \dots < t_k \leq 1$, the changes $[W(t_2) - W(t_1)]$, $[W(t_3) - W(t_2)]$, $[W(t_4) - W(t_3)]$, ..., $[W(t_k) - W(t_{k-1})]$ are independent multivariate Gaussian with $[W(s) - W(t)] \sim N(0, s - t)$;
- c) For any given realization, $W(t)$ is continuous in t with probability 1.

Note that, although $W(t)$ is continuous in t , it can not be differentiated using standard calculus; the direction of change at t is likely to be completely different from that $t + \Delta t$, no matter how small one make Δ .

2.2. Integrated Processes

A random walk is an example of a class of trending processes known as Integrated Processes. To give a precise definition of integrated processes, we first define I(0) processes. An I(0) process is a strictly stationary process whose long-run variance is finite and positive as we indicate it in preceding part by Stationary Conditions (second criteria). We allow I(0) processes to have possibly nonzero means. Therefore, an I(0) process x_t can be written as

$$x_t = \alpha + u(t)$$

where $\{u_t\}$ is zero-mean stationary with long-run variance.

The definition of integrated processes follows from the definition of I(0) processes. Let Δ be the difference operator, so, for a sequence $\{x_t\}$,

$$\Delta x_t = (1 - L)x_t = x_t - x_{t-1} \tag{2.4}$$

⁴ Hamilton, J.D, 1994, *Time Series Analysis*, Princeton University Press

$$\Delta^2 x_t = (1 - L)^2 x_t = (x_t - x_{t-1}) - (x_{t-1} - x_{t-2})$$

and so on.....

As is clear with random walks, the variance of an I(1) process increases linearly over time. Thus if the process had started in the infinite past, the variance would be infinite. To focus on I(1) processes with finite variance, we assume that the process began in the finite past, and without loss of generality we can assume that the starting date is $t = 0$. Since an I(0) process can be written as (2.4), we can write $\{x_t\}$ in levels as follows:

$$x_t = x_0 + \alpha t + (u_1 + u_2 + \dots + u_t) \quad (2.5)$$

Where $\{u_t\}$ is zero-mean I(0). So the specification of the levels process $\{x_t\}$ must include an assumption about initial condition. Unless otherwise stated, we assume throughout that $E(x_t) < \infty$. So x_0 can be random.

As we can easily see, the mean in I(0) process is the trend in I(1). As is clear from (2.5), an I(1) process can have a linear trend, (αt) . This is a consequence of having allowed I(0) processes to have a nonzero mean α . If $\alpha = 0$, then the I(1) process has no trend and is called a driftless I(1) process, while if $\alpha \neq 0$, the process is called an I(1) process with drift. Evidently, an I(1) process with drift can be written as the sum of a linear trend and a driftless I(1) process.

An I(1) process has two other names. It is called a Difference Stationary (DS) process as we explained in 2.1.1.b because the first difference of this process is stationary. The second name of an I(1) process is Unit-Root process. To see why it is so called, consider a model

$$(1 - \rho L)x_t = \alpha + u_t$$

where u_t is zero-mean I(0). It is an autoregressive model with possibly serially correlated errors represented by u_t . If the autoregressive root ρ is unity, then the first difference of x_t is I(0), so $\{x_t\}$ is I(1).

2.2.1. Why is it important to know if the Process is I(1)?

First, it matters a great deal in forecasting. To make the point, consider the following simple AR(1) process with trend:

$$x_t = \alpha + \beta.t + y_t$$

$$y_t = \rho y_{t-1} + \varepsilon_t \quad \text{where } \{\varepsilon_t\} \sim N(0, \sigma) \text{ is white noise.}$$

The s -period ahead prevision or forecast of y_{t+s} conditional on $(y_t, y_{t-1}, \dots)$ can be written as

$$E(y_{t+s} | y_t, y_{t-1}, \dots) = \alpha + \beta.(t+s) + E(y_{t+s} | y_t, y_{t-1}, \dots)$$

Time Series is concerned with estimation of relationships among groups of variables, each of which observed at a number of consecutive points in time. The relationships among these variables may be complicated; in particular, the value of each variable may depend on the values taken by many others in several previous time periods. In consequence, the effect that a change in one variable has another effect depending upon the time horizon that we consider. It is easy to imagine examples in which a change in one quantity has little or no effect on another at first and a substantial effect later. Alternatively, a variable may have a substantial effect on another for a time, but that effect may eventually die out.

It is useful, therefore, to distinguish what are often called “short-run” relationships (those holding over a relatively short period) from “long-run” relationships. The former relate to links that do not persist. The long-run relationships are closely linked to the concepts of equilibrium relationships.

2.2.2 Equilibrium Relationships and the Long-Run

An equilibrium state is defined as one in which there is no inherent tendency to change. A disequilibrium is any situation that is not an equilibrium and hence characterizes a state that contains the seeds of its own destruction. An equilibrium state may or may not have the property of either local or global stability; thus, it may or may not be true that the system tends to return to the equilibrium state when it is perturbed. However, we

generally consider only stable equilibria, since unstable equilibria will not persist given that there are stochastic shocks to the series. That is, equilibria are states to which system is attracted, other things being equal. It may also be possible in some circumstances to view the forces tending to push the system back into equilibrium as depending upon the magnitude of the deviation from equilibrium at a given point in time.

Equilibrium may be either general or partial. In the latter case, a given system (signal source) is viewed as having attained equilibrium in spite of the fact that we have not taken account of the feedback from other sources. In both cases, an equilibrium relationship is expressed through a function $f(x_1, x_2, x_3, \dots, x_n) = 0$, which describes the relationships that hold among the n variables x_1 to x_n when the system is in equilibrium. The concept “*long-run equilibrium*” is also used to denote the equilibrium relationship to which a system converges over time. Over finite periods of time, the long-run or equilibrium relationships may fail to hold, but they will eventually hold to any degree of accuracy if the equilibrium is stable, and if the system does not experience further shocks from outside. Expressed differently, a long-run equilibrium relationship entails a systematic co-movement among system variables which a system exemplifies precisely in the long-run; in general within the notations of economic theory literature one writes equations representing such co-movements without time subscripts as $x_1 = \beta x_2$ to denote a linear long-run relation between x_1 and x_2 .

In our research context, we can say that : Even if shocks to a system are constantly occurring so that the neural system is never in equilibrium, the concept of long-run equilibrium may nonetheless be useful. The present is the long-run outcome of the distant past and, as will be made precise below, a long-run relationship will often hold “on average” over time. Moreover, a stable equilibrium has the property that a given deviation from the equilibrium becomes more and more unlikely as the magnitude of the deviation is greater, so the one may be reasonably connecting variables and this long-run relationship is within certain bounds.

2.2.3 Stationary and Equilibrium

The statistical concept of equilibrium centres on that of stationary process. A substantial body of methods is developing around the statistical features of equilibrium relationships among time-series process, and the concepts of stationarity and particular forms of non-stationarity are crucial to these methods.

If a particular relationship such as $x_1 = \beta x_2$ emerges as the voxel(s) system is allowed to settle down, this will describe an equilibrium to a theorist. In actual time series, however, the relation $x_{1t} = \beta x_{2t}$ may never be observed to hold. Consequently, we look for ways of characterizing the relationships that can be observed to hold between x_{1t} and x_{2t} .

In general, an equilibrium relationship is said $f(x_1, x_2) = 0$ holds between two variables x_1 and x_2 if the amount $\varepsilon_t \equiv f(x_{1t}, x_{2t})$ by which actual observations deviate from this equilibrium is a median-zero stationary process. That is, the “error” or discrepancy between outcome and postulated equilibrium has a fixed distribution, centered on zero, that does not change over time. This error cannot therefore grow indefinitely; if it did, the relationship could not have been an equilibrium one since the system is free to move ever further away from it. It may be difficult to distinguish in finite samples between an ever-growing discrepancy in an hypothesized equilibrium relationship and a random fluctuation.

Given the characterization above, the short-run discrepancy ε_t in an equilibrium relationship must have no tendency to grow systematically over time. However, since this error represents shocks that are constantly occurring and effecting neighbourhood voxel variables.

This definition of an equilibrium relationship holds automatically when applied to series that are themselves stationary. For any two stationary series $\{x_{1t}\}$ and $\{x_{2t}\}$, irrespective of any substantive relationship between these two alone, a difference of the form $\{x_{1t} - bx_{2t}\}$ must be a stationary series for any b . Thus, whether or not there exists a

non-zero b which describes a true equilibrium relationship, corresponding to a non-zero derivative between x_1 and x_2 , any arbitrarily chosen b will meet the statistical equilibrium condition.

2.2.4 Equilibrium and the Specification of Dynamic Models

If there exists a stable equilibrium: $x_1 = \beta x_2$, the discrepancy $\{x_{1t} - \beta x_{2t}\}$ evidently contains useful information since on average the system will move towards that equilibrium if it is not already there. In particular, $\{x_{1t-1} - \beta x_{2t-1}\}$ represents the previous disequilibrium. Suppose the equilibrium relationship is between a variable $\{y_t\}$ to be modelled and some series $\{z_t\}$ which is exogenous in an appropriate sense. If we let $x_{1t} = y_t$ and $x_{2t} = z_t$ to distinguish their status, and denote the equilibrium by $y = \beta z$, then the innovation or error, $\{y_t - \beta z_t\}$ should be a useful explanatory variable for the next direction of movement of y_t . In particular when $y_t - \beta z_t$ is positive, y_t is too high relative to z_t , and on average we might expect a fall in y in future periods relative to its trend growth. The term $\{y_{t-1} - \beta z_{t-1}\}$, called an *error-correction mechanism*, is included sometimes in dynamical regressions.

The true parameter β characterizing the relationship is not known in general. This need not prevent error-correction mechanism from being useful, however, since unknown parameter can either be estimated separately in a priori analysis or estimated in the course of modelling the variable of interest. Moreover, the general error-correction mechanism can be shown to be equivalent to various other transformations of a general linear model incorporating past values of both the variable of interest and explanatory variables. A particular advantage of the error-correction mechanism is that the extent of adjustment in a given period to deviations from long-run equilibrium is given by the estimated equation without any further calculation.

The practice of exploiting information contained in the current deviation from an equilibrium relationship, in explaining the path of a variable, has benefited from the

formalization of the concept of *co-integration* by Engle and Granger (1987), we will give its formal definition in part 2.2.7. The informal definition of statistical equilibrium discussed above is based upon a special case of the definition of *co-integration*. Further the practice of modelling *co-integrated series* is closely related to error-correction mechanisms.

A series that is tending to grow over time cannot be stationary (although it may possibly be stationary around some deterministic trend explained in part 2.1), but the changes in that series might be. To take a mechanical example, if an object has a fixed average position around which it moves, always returning after some interval to this position like a randomly perturbed weight at the end of a spring, then its displacement may be a stationary series. An object that has no such fixed position may nevertheless have a velocity (the change in position per unit time), or acceleration, that is stationary. For example, if the object is moving ever further from its point of origin, but with velocity fluctuating around some fixed positive mean according to a fixed distribution function, then the velocity of the object is a stationary series.

From the preceding part we know that a series is said to be integrated of order 1 (I(1)) if, although it is itself non-stationary, the changes in this series form a stationary series. It is said to be integrated of order 2 (I(2)) if, although the changes are non-stationary, the changes in the changes form a stationary series. In other words, if the series must be differenced exactly k times to achieve stationarity, then the series is I(k), so that a stationary series is I(0).

We can now consider the concept of co-integration, its relation to the definition of long-run equilibrium between series given above, and its use as part of a statistical description of the behaviour of time series that satisfy some equilibrium relationship. A simple example concerns two series, each of which integrated of order 1. Assume that a long-run equilibrium relationship holds between them, and that is linear: $x_1 = \beta x_2$. Then $(x_1 - \beta x_2)$ must be equal to zero in equilibrium and the series $\{x_{1t} - \beta x_{2t}\}$ has a constant unconditional mean of zero. This need not imply that $\{x_{1t} - \beta x_{2t}\}$ is stationary: as we explained the conditions to be stationary in part 4.1., the variance of $\{x_{1t} - \beta x_{2t}\}$ might

be non-constant, for example. The definition of *co-integration* given by Engle and Granger (1987) does however require stationarity of the deviation $\{x_{1t} - \beta x_{2t}\}$. When stationarity does hold, we say that x_1 and x_2 are co-integrated (1,1) and denoted CI(1,1); that is, they are each integrated of order 1, and there exists some linear combination $\{x_{1t} - \beta x_{2t}\}$ which is integrated of an order one lower than the components (i.e. I(0) here). If $\{x_{1t} - \beta x_{2t}\}$ has a constant unconditional mean but is not stationary, then we may still want to say that an equilibrium relationship holds; the series will not, however, fit the strict Engle and Granger definition of co-integration, which requires that some linear combination be stationary.

A substantive long-run equilibrium relationship is something from which the variables involved can deviate, but not by an ever-growing amount. That is, the discrepancy or error in the relationship cannot be integrated of any order greater than zero. Series integrated of strictly positive orders which are linked by such an equilibrium relationship must, therefore, be co-integrated with each other. In the example just given, the fact that the integrated series x_1 and x_2 move together in the long run is reflected in the fact that they are co-integrated; a linear relation yields a stationary deviation.

2.2.5 Properties of Integrated Processes

A knowledge of the fundamental properties of integrated processes is essential for an understanding of tests for both non-stationary and the existence of long-run equilibrium relationships. We deal with the issue of spurious regressions and show how a consideration of the theory of integrated processes helps us to understand the behaviour of standard estimators in models involving non-stationary data. Much conventional asymptotic theory for least-squares estimation (standard proofs of consistency and asymptotic normality of Ordinary Least Squares - OLS estimators) assumes stationarity of the explanatory variables, possibly around a deterministic trend. Nonetheless, regression methods have often appeared to be effective when analysing such series, and it was not clear that methods developed for stationary series would not be valid elsewhere.

Some examples of what can emerge when standard regression techniques are used with non-stationary data were re-emphasized by Granger and Newbold, who considered the following data generation process:

$$\begin{aligned} y_t &= y_{t-1} + u_t, \quad u_t \sim i.i.d \quad (0, \sigma_u^2) \\ x_t &= x_{t-1} + v_t, \quad v_t \sim i.i.d \quad (0, \sigma_v^2) \\ E(u_t v_s) &= 0, \forall v, s \quad ; \quad E(u_t u_{t-k}) = E(v_t v_{t-k}) = 0, \forall k \neq 0 \end{aligned}$$

That is, x_t and y_t are uncorrelated random walks. Since x_t neither affects nor affected by y_t , one would hope that the coefficient β_1 in the regression model

$$y_t = \beta_0 + \beta_1 x_t + \varepsilon_t$$

would *converge in probability (plim)* to zero, reflecting the lack of a relation between the series, and that the coefficient of determination (R^2) from this regression would also tend to zero. However, this is not the case. Regression methods detect correlations, and in non-stationary series spurious correlations may persist in large samples despite the absence of any connection between underlying series. If two time series are each growing, for example, they may be correlated even though they are increasing for entirely different reasons and by increments that are uncorrelated. Hence a correlation between integrated series cannot be interpreted in the way that it could be if it arose among stationary series.

Trends and Random Walks:

As explained in part 2.1, one potential solution suggested for dealing with integrated series was to assume that the source of non-stationarity could be captured by, or approximated by, a deterministic function of time. If this were so, it would be possible to break up an integrated series into a deterministic component, and a stationary series of deviations from its “trend”. However, time trends would appear to be statistically significant in models where they should not be. Moreover, deterministic trends did not solve the spurious regression problem.

Consider a series $\{y_t\}$ which is generated according to the random walk

$$y_t = y_{t-1} + \varepsilon_t, \quad \varepsilon_t \sim i.i.d \quad (0, \sigma^2)$$

Without knowing this data generation process, an investigator faced with such series might decide to attempt to deal with the apparent non-stationarity by de-trending; that is, including a time trend in a regression equation or by removing the fitted values from regression on time from the series. The investigator might therefore use the regression model

$$y_t = c + \gamma t + u_t$$

But, when $c = \gamma = 0$ from the equation cited above, $\hat{\gamma}$ has a degenerate limiting distribution at 0 (as in a stationary model with a trend).

Since the spurious regression problem between integrated series remains with deterministically de-trended series, inclusion of a time trend is not a solution.

Before we consider testing for integration in time series, we must first define orders of integration and consider some of the properties that integrated series usually display.

*Definition*⁵: A series with no deterministic component and which has a stationary and invertible autoregressive moving average (ARMA) representation after differencing d times, but which is not stationary after differencing only $(d-1)$ times, is said to be integrated of order d , denoted $x_t \sim I(d)$.

The properties of series integrated of strictly positive orders differ substantially from those of $I(0)$ series. Consider a series containing a single unit root:

$$y_t = \rho y_{t-1} + u_t; \quad y_0 = 0, \quad u_t \sim I(0)$$

Or after integrating,

$$y_t = S_t \quad \text{where } S_t \equiv \sum_{i=0}^{t-1} \rho^i u_{t-i}.$$

⁵ Banerjee, A., Dolado J.J., Galbraith, W.J., and Hendry D., 1993 “Co-Integration, error correction, and the econometric analysis of non-stationary data” Oxford University Press

If $|\rho| \geq 1$, y_t is non-stationary, and if $\rho = 1$, it is integrated of order 1 (i.e. I(1)) since y_t is then the sum of all previous errors $\{u_j\} \ j=1,2,\dots,t$. The sequence $\{u_t\}$ need not be an innovation sequence; u_t may itself follow a stationary ARMA(p,q) process, for example.

For comparing Integrated Process, assume two Data Generating Processes (DGP) :

$$y_t = \rho y_{t-1} + u_{1t} ; \quad |\rho| < 1 ; \quad u_{1t} \sim IID(0, \sigma_1^2) \quad (2.6)$$

And

$$y_t = y_{t-1} + u_{2t} ; \quad y_0 = 0 ; \quad u_{2t} \sim IID(0, \sigma_2^2) \quad (2.7)$$

Table 1. The comparison of I(0) and I(1) Processes

	DGP(2.6) I(0)	DGP(2.7) I(1)
Variance	Finite $(\sigma_1^2(1-\rho^2)^{-1})$	Unbounded Grows as $t\sigma_2^2$
Conditional Variance	σ_1^2	σ_2^2
Autocorrelation function at lag i	$\rho_i = \rho^i$	$\rho_i = \sqrt{1 - \left(\frac{i}{t}\right)} \rightarrow 1 \forall i \text{ as } t \rightarrow \infty$
Expected time between crossings of y=0	Finite	Infinite
Memory	Temporary	Permanent

2.2.6 Testing For Unit Root

Since an I(1) series becomes stationary upon being differenced once, it must contain one unit root. For example, if we take a random walk as DGP, then we can immediately derive that its first difference is stationary. If by contrast the underlying data generating process is

$$y_t = \rho_1 y_{t-1} + u_{1t} ; \quad |\rho_1| > 1; \quad \text{then we have}$$

$$y_t - y_{t-1} \equiv \Delta y_t = (\rho_1 - 1)y_{t-1} + u_{1t} \quad (2.8)$$

From (2.8) it is clear that Δy_t is no longer stationary: it depends not only the stationary process u_{1t} , but also upon the non-stationary process y_{t-1} (since $\rho_1 - 1 > 0$). Hence an AR(1) process with a coefficient of 1 is I(1), but same process with a coefficient of 1.01 is not, since differencing will not reduce this process to stationarity.

Consider the simplest data generation process within which we can discuss tests for unit roots:

$$y_t = \rho y_{t-1} + u_t ; \quad \text{where } u_t \sim i.i.d(0, \sigma_u^2) ; \quad (2.9)$$

$$y_0 = 0$$

If one were testing the true hypothesis $H_0 : \rho = \rho_0$ for $|\rho_0| < 1$, the test would be easily performed. Running the regression (2.9), the t-statistic $\frac{(\hat{\rho} - \rho_0)}{SE(\hat{\rho})}$ has, asymptotically, a standard normal distribution and can be compared with tables of significance points for N(0,1).

For $\rho_0 = 1$, however, this result no longer holds. The distribution of the test statistic just given is not asymptotically normal, or even symmetric. Tables of critical values have been tabulated by D.A. Dickey and are reported in e.g. Fuller (1976). It is instructive to examine these in detail, and are recorded as Tables A.2 and A.3.

The critical values in Fuller's tables pertain to each of three different models: It is important to note at the outset that, as in many other instances, the distributions of test statistics obtained depend not only on the data generation process, but also on the model with which we investigate it. We consider three models:

By their ordering we call these as Case 1 ; Case 2 ; Case 3 to read both critical values of Table A.2 and Table A.3.

$$y_t = \rho_a y_{t-1} + u_t \quad (2.10)$$

$$y_t = \mu_b + \rho_b y_{t-1} + u_t \quad (2.11)$$

$$y_t = \mu_c + \gamma_c t + \rho_c y_{t-1} + u_t \quad (2.12)$$

The null hypothesis is that $H_0 : \rho_i = 1$ for $i = a, b, c$

For example, consider model (2.11). A t-statistic of +1.00 would not lead to rejection of the null against an explosive alternative if we were applying N(0,1) tables; by Table A.3 **case 2**, however, the test rejects at the 5 percent level (or even 1 percent level) because the probability of the statistic exceeding even 0.60 is only 0.01. By contrast, a value of -2.50, which would lead to rejection of H_0 using standard normal tables, can no longer be used to infer that $H_0 : (\rho = 1)$ is false against a stationary alternative at the % 5 level.

2.2.6.1 Testing for Unit Root in General Dynamical Models for the Process of Interest:

The first of the methods for allowing richer dynamics in the DGP of the process of interest, $\{y_t\}$, was developed concurrently with the test that we already described for a unit root in AR(1) model, and is reported in Fuller (1976). These more general methods yield test statistics that have the same limiting distributions as those already discussed. Hence, we may use the last rows of Table A.2 case 1 and case 3 or Table A.3 case 1 and case 3 for inference with these statistics in large sample, but in small samples percentage

points of their distributions will not general be the same as those applicable under the strong assumptions of the simple Dickey-Fuller model.

When y_t follows an AR(p) process,

$$y_t = \sum_{i=1}^p \rho_i y_{t-i} + \varepsilon_t \quad (2.13)$$

A test can be constructed with the regression model :

$$y_t = \rho y_{t-1} + \sum_{i=1}^{p-1} \gamma_i \Delta y_{t-i} + u_t \quad (2.14)$$

The coefficient ρ is used to test for a unit root, and $T(\hat{\rho}-1)$ and $\frac{(\hat{\rho}-1)}{SE(\hat{\rho})}$ have limiting distributions tabulated in Tables A.2. case 1 and Table A.3.case 1 for $T \rightarrow \infty$. Moreover, just in the case of an AR(1) process, we can extend this regression model to allow for the possibility that the data generation process contains a constant (drift) term or a deterministic time trend. Again, for suitably modified regression models, the asymptotic distributions of the statistics based on $\hat{\rho}$ are those given in Tables A.2 case 2 and 3 and Table A.3 case 2 and 3 for $T \rightarrow \infty$. These procedures are called “Augmented” Dickey-Fuller (ADF) tests.

The aim in modifications such as these to simpler form of the Dickey-Fuller test is to use lagged changes in the dependent variable to capture autocorrelated omitted variables which would otherwise, by default, appear in the (necessarily autocorrelated) error term. With additional lagged terms it will be possible, if the DGP has the form of (2.13), to produce (2.14) in which asymptotically the error terms are white noise, because the nuisance parameters are known asymptotically and the terms involving them may be removed from the error term. Moreover, the asymptotic distribution of the coefficient on the y_{t-1} in (2.14) is not affected by the inclusion of the additional term Δy_{t-i} terms. If y_t is I(1), the differenced terms are all I(0) and appropriate scaling ensures that the variance-covariance matrix is asymptotically block-diagonal.

By allowing the DGP to take the form (2.13) rather than the much more restrictive AR(1) form (4.10 or 4.11 or 4.12), we expanded the class of models to which we can validly apply unit-root tests of this type. Note that, as it generally be the case that p is unknown even where y_t is strictly an AR(p) process, we have to predict the value of p by various tests (see Hall 1990).

The next step is to attempt to extend further the class of series to which we can apply such tests, ideally in such a way as to allow exogenous variables to enter the process as well. Said and Dickey (1984) provide a test procedure valid for a general ARMA process in the errors; Phillips(1987a) and Perron and Phillips (1988) offer a still more general procedure.

The method of Said and Dickey involves approximating the true process by an autoregression in which the number of lags increases with sample size.

Begin by assuming that the data generation process follows:

$$y_t = \rho y_{t-1} + u_t$$

$$u_t + \sum_{i=1}^p \alpha_i u_{t-i} = e_t + \sum_{j=1}^q \theta_j e_{t-j}$$

$$e_t \sim \text{i.i.d}(0, \sigma_e^2)$$

So that the error term in the autoregression follows an ARMA(p,q), presumed to be stationary and invertible. The DGP can be written as

$$\Delta y_t = (\rho - 1)y_{t-1} + \sum_{i=1}^k \alpha_i \Delta y_{t-i} + v_t \quad (2.15)$$

Where k is large enough to allow a good approximation to the ARMA(p,q) process $\{u_t\}$, so that $\{v_t\}$ is approximately white noise. The null hypothesis is again that $\rho = 1$. Said and Dickey show that the test is valid in spite of the facts that p and q are unknown. OLS estimation of the model (2.15) is proven to yield a consistent estimator of $(\rho - 1)$; the test can be then based on the t-type statistic, $\frac{(\hat{\rho} - 1)}{SE(\hat{\rho})}$, using Table A.3 **case 1**. Clearly,

the form of the regression implied by the Said-Dickey test is precisely the same as that of the augmented Dickey-Fuller test.

Note that, Monte Carlo studies of test power in models with autocorrelated error process, described by Dickey et al.,(1986), suggest that the empirical levels of the $T(\hat{\rho} - 1)$ statistics tend to be farther from the nominal test levels than those of the t-type statistics, $\frac{(\hat{\rho} - 1)}{SE(\hat{\rho})}$. Dickey et al.,(1986) therefore suggest to use of the t-type statistics in these cases.

2.2.7 Co - Integration

In part 2.1 we briefly discussed use of the word “equilibrium” in economics. The idea that the variables hypothesized to be linked by some theoretical economic relationship should not diverge from each other in the long-run is a fundamental one. As in economics, like the separation of such variables in short-run because of seasonal effects, also in brain research we can view such variables may drift apart in the short – run or because instantaneous effects, but if they were to diverge without bound, an equilibrium relationship among such variables could not be said to exist. The divergence from a stable equilibrium state must be stochastically bounded and, at some point, diminishing over time. “Co – Integration” may be viewed as the statistical expression of the nature of such equilibrium relationships.

The concept of co – integration is a powerful one because it allows us to describe the existence of an equilibrium, or stationary, relationship among two or more time-series, each of which is individually non – stationary. That is, while the component time-series may have moments such as means, variances, and covariances varying with time (refer the conditions explained in part 42.1), some linear combination of these series, which defines the equilibrium relationship, has time – invariant linear properties.

Definition (Engle and Granger (1987)): The components of the vector x_t are said to be co-integrated of order d, b , denoted $x_t \sim CI(d, b)$, if

- (i) x_t is $I(d)$

(ii) there exists a non-zero vector α such that $\alpha'x_t \sim I(d-b)$, $d \geq b > 0$. The vector α is called the co-integrating vector.

If there exist exactly r linearly independent co –integrating vectors with $r \leq n-1$, then these can be gathered into an $n \times r$ matrix α . The rank of α will be r and is called the *co – integration rank*.

In order to illustrate the preceding discussion, consider that:

Two time series $\{x_t\}$ and $\{y_t\}$ are each integrated of order 1 and evolve according to following data – generation process:

$$x_t + \beta y_t = u_t \quad \text{where} \quad u_t = u_{t-1} + \varepsilon_{1t} \quad (2.16)$$

$$x_t + \alpha y_t = v_t \quad \text{where} \quad v_t = \lambda v_{t-1} + \varepsilon_{2t} \quad \text{with} \quad |\lambda| < 1 \quad (2.17)$$

and $(\varepsilon_{1t}, \varepsilon_{2t})'$ is i.i.d as bivariate normal with

$$E(\varepsilon_{1t}) = E(\varepsilon_{2t}) = 0 ;$$

$$\text{var}(\varepsilon_{1t}) = \sigma_{11} ; \quad \text{var}(\varepsilon_{2t}) = \sigma_{22} ; \quad \text{cov}(\varepsilon_{1t}, \varepsilon_{2t}) = \sigma_{12}$$

Solving for x_t and y_t from the above system with $\alpha \neq \beta$ gives

$$x_t = \alpha(\alpha - \beta)^{-1} u_t - \beta(\alpha - \beta)^{-1} v_t$$

$$y_t = -(\alpha - \beta)^{-1} u_t + (\alpha - \beta)^{-1} v_t$$

Since $\{u_t\}$ is a random walk and $\{x_t\}$ and $\{y_t\}$ depend linearly on $\{u_t\}$, these may therefore be classified as $I(1)$ variables. Nonetheless, $\{x_t + \alpha y_t\}$ is $I(0)$ because $v(t)$ is stationary in (2.17). The vector $[1 : \alpha']$ is the co-integrating vector and $x + \alpha y$ is the equilibrium relationship. In the long-run, the variables move towards the equilibrium $x + \alpha y = 0$, recognizing that this relationship need not be realized exactly even $t \rightarrow \infty$.

2.3 Application of Non-Stationary Analysis to the Clinical Alzheimer and Epilepsy EEG data.

The number of measurements given at one instant of time is 19; the standard 10–20 system of clinical EEG recordings is employed. The observation data can be supplied upon to request.

We think that, if the EEG data which we examine has Non-Stationary behaviour, from the theoretic base explained in 2.1, it may be in two different formats:

$$1. y_t = \alpha + \beta t + u_t \Rightarrow u_t \sim ARMA(p, q) \text{ or} \quad (2.18)$$

$$\delta(L)u_t = \mu(L)\varepsilon_t \text{ where } \varepsilon_t \sim N(0, R)$$

$$2. \phi(L)\Delta y_t = \zeta(L)\varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, R) \quad (2.19)$$

In the light of these two different structures, we will search and analyze EEG signals or recordings individually (one by one) from 19 different channels (that means: for any person we test at least 19 EEG signals) to determine whether there exists Non-Stationarity in whole or any subinterval of EEG data taken from that channel or that region and we do this analysis for all individuals. After we examine the nature of EEG data in every channel or region or source and if we obtain Non-Stationary process hidden in these EEG data, then we will examine whether it affects other regions or sources by Co-integration theory and its test methods.

For determining the existence of Non – Stationary, we use ADF statistic tests explained in part 2.2.6.1.

- i). First of all we will distinguish which of the two equations (2.18) or (2.19) characterizes our EEG data.
- ii.) Secondly, we assume that there may be a time trend and drift $(a + bt)$ in these EEG data. And construct our Null Hypothesis according to this assumption. We apply Non-standard t-statistics by OLS.

iii.) Thirdly, we explore that, whether the innovations within the data are i.i.d noise or have Autoregressive properties (shown with u_t in the first equation above). If they have Autoregressive character, then we must first find its order by using Theorem of *Phillips – Perron* and *Hannan Test* and eliminate them by using the method of *Said and Dickey* to make A.D.F Tests: This method is called “whitening the noise”. After this elimination we can make Non-Stationary tests.

When we realized the first step, we find that the EEG data observed (generated by human brain) has no drift and independent time trend. So we must focus on (2.19). As we look for Unit Root in the EEG data, we can write its open form as follows.

$$y_t = \alpha + \beta t + \rho y_{t-1} + \sum_{i=1}^p \gamma_i (y_{t-i} - y_{t-i+1}) + \varepsilon_t \quad \text{where} \quad \varepsilon_t \sim N(0, \sigma) \quad (2.20)$$

$$\text{or} \quad \Delta y_t = \alpha + \beta t + (\rho - 1)y_{t-1} + \sum_{i=1}^p \gamma_i (y_{t-i} - y_{t-i+1}) + \varepsilon_t \quad \text{where} \quad \varepsilon_t \sim N(0, \sigma)$$

Now as we indicated, we applied this equation the A.D.F tests to explore whether it contains Non-Stationarity or not. One can regard the results from the OLS regressions cited in the tables. We applied this test method (explained in 2.2.6) to every EEG signal through every channel for every person . You can view some of them from the tables.

From the statistical tables below, you can easily see that:

1. For the samples of EPILEPSY data taken by various electrodes, for the part of 512 sample (2 sec), there is exactly one unit root in the time series.

However, this unit root disappears when the process gets longer. In the serie for 2048 sample, there is not unit root in the EPILEPSY data, and the series become Stationary. This is very interesting case, because in EEG series, it occurs a transition from Random Walk to Stationary AR(p) process. This means that EEG in fact contains both a random walk process and stationary AR(p) process in its nature.

1.b. The Random walk occuring within 2 sec in any of the 19 zones, diffuse very quickly to other zones, this can be seen, from the differences in lags of the series taken from

different electrodes. Hence, we can exactly derive the origin zone of the Random Walk process.

1.c. After 2 sec, these random walk process all transform to stationary AR(p) process. This is very interesting. Physiologically, I can conclude that, the perturbation of neurons from the preceding crises lowers the transmitting performance of certain zones. This exactly cause the shocks in (great innovations) in the series when compared with normal or Alzheimer Data.

2. In Alzheimer data, as seen from Table A.13 there is no Random Walk process pattern captured. Alzheimer data is Stationary like a normal individual.

3. All EEG time series are stationary within whole data string. That is, when series with great observation is analysed, it would not found any unit root. With 76000 samples all series are stationary.

All the OLS result tables Tables A.1; A.4; A5; A6 and A7 shows that: there is Unit Root in Epilepsy data taken from different individual's different brain zones. That means: Epilepsy is strictly characterized by Unit Root or Non-Stationary signal generator regions.

After this finding, we will explore the relation between the behaviours of such “diseased regions”. Table A.8 and A.9 indicates this result. There is exactly co – movement or Co-Integration relation between the regions that generates Unit Root. This is very nice result. Because, this result brings us to Granger's Causality Theorem (1987); we can exactly find which one is the pioneer or trigger of Non-Stationary Data generation.

Now we extend this Co-Integration analysis and we examine the causality between these two regions. Causality Theorem is introduced by Engle and Granger (1987). If we can estimate a Co-Integration vector that links O1_AV and O2_AV each other then we can estimate which affects the other in both Long-Run and Short-Run. To analyze these effects we use Vector Error Correction Mechanism (VECM) proposed by Engle and Granger (1987). We know from the Table A.6 and Table A.7 that :

$$\Delta(O1_AV)_t = (\rho - 1)(O1_AV)_{t-1} + \sum_{i=1}^{16} \gamma_i \Delta(O1_AV)_{t-i} + \varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma) \quad (2.21)$$

$$\Delta(O2_AV)_t = (\rho - 1)(O2_AV)_{t-1} + \sum_{j=1}^{13} \gamma_j \Delta(O2_AV)_{t-j} + \varepsilon_t \quad \text{where } \varepsilon_t \sim N(0, \sigma)$$

From these Tables we test hypothesis that : $(H_0 : \rho = 1)$ against $(H_1 : \rho < 1)$. We can easily infer from using Table A.3 that, both O1_AV and O2_AV has Non-Stationary characters. That means, for both series $(H_0 : \rho = 1)$ is accepted with % 95 significance level.

2.3.1 Tests for Co-Integration and Determination of Co-Integration Vector If It Exists.

From the theoretical explication of Co-Integration, we will explore a link between two Non-Stationary data generated by regions O1_AV and O2_AV as follows:

$$\beta_1(O1_AV) + \beta_2(O2_AV) \sim I(0) \quad (\text{Co-Integration relation})$$

That is :

$$\beta_1(O1_AV) = \alpha - \beta_2(O2_AV) + u_t \quad (2.22)$$

Or

$$(O1_AV)_t = \alpha + b(O2_AV)_t + \hat{u}_t \quad (2.23)$$

We search a normalized vector $b = \left[1; \frac{-\beta_2}{\beta_1} \right]$ called Co-Integration vector. This Co-

Integration vector shows us the Long-Run relation between O1_AV and O2_AV.

From the regression results shown in Table A.9, the unknown coefficients in (2.23) determined with a significance level % 95 as follows:

$\alpha = -3.052537$ and vector $b = (1: 0.570657)$. That means from the equivalent equation (2.22) that $\beta_1 = 1$ and $\beta_2 = -0.570657$

Now, we have another problem to decide whether Co-Integration exists or not? That is, in the (2.23), does u_t ARMA(p,q) or I(h) character, where h is not zero ? Because from the definition of Co-Integration given in part 2.2.7 and also given with equation $\beta_1(O1_AV) + \beta_2(O2_AV) \sim I(0)$; we have to explore the Stationarity of u_t (residuals) which can be written as follows from the (2.23):

$$(O1_AV) - \alpha - b(O2_AV) = \hat{u}_t$$

$$\Rightarrow (O1_AV) - (-3.052537) - (0.570657).(O2_AV) = \hat{u}_t$$

2.3.1.1 Testing the Non-Stationarity of Residuals ($\hat{u}_t$) of Co-Intergation relation ?

$$\Delta \hat{u}_t = \chi + \delta + \rho.u_{t-1} + \sum_{k=1}^z \theta_k . \Delta \hat{u}_{t-k} + \varepsilon_t \text{ where } \varepsilon_t \text{ is i.i.d and } \varepsilon_t \sim N(0, \sigma)$$

The null hypothesis of no-cointegration implies that $\hat{u}_t$ is I(1).

From the Table A.10 we test hypothesis that : ($H_0 : \rho = 0$) using the Tables of Phillips-Oularis given in Table A.14 (case 1). One can easily see that, we accept that residuals, $\hat{u}_t$ is Stationary with confidence level %95. The estimated equation will be,

$$\Delta \hat{u}_t = \rho.u_{t-1} + \sum_{k=1}^8 \theta_k . \Delta \hat{u}_{t-k} + \varepsilon_t \quad (2.24)$$

and in the Table A.10 , $\hat{u}_t$ is shown by “CO”.

In Table A.14 we have three different cases: Case 1 ; Case 2; Case 3. These cases varies because the type of equations estimated. These types can be given as follows:

$$1. \Delta \hat{u}_t = \rho.u_{t-1} + \sum_{k=1}^8 \theta_k . \Delta \hat{u}_{t-k} + \varepsilon_t \quad (\text{Case 1}) \text{ “demeaned and detrended”}$$

$$2. \Delta \hat{u}_t = \lambda + \rho.u_{t-1} + \sum_{k=1}^8 \theta_k . \Delta \hat{u}_{t-k} + \varepsilon_t \quad (\text{Case 2}) \text{ “meaned and detrended”}$$

$$3. \Delta \hat{u}_t = \lambda + \eta.t + \rho.u_{t-1} + \sum_{k=1}^8 \theta_k . \Delta \hat{u}_{t-k} + \varepsilon_t \quad (\text{Case 3}) \text{ “meaned and trended”}$$

As because we find a Co-Integration vector and tested the Stationarity of residuals, now we can explore whether the regions affects each other or not by using VECM?

1. Does O2_AV affect O1_AV in the Long-Run and/or in the Short-Run?

$$\Delta(O1_AV)_t = \omega + \sum_{i=1}^p a_i \Delta(O1_AV)_{t-i} + \sum_{j=1}^q e_j \Delta(O2_AV)_{t-j} + r.\hat{u}_{t-1} \quad (2.25)$$

This equation questions that: Does the first difference of Non-Stationary data generated by region or source O1_AV depend on its own history and/or the history of data generated by region or source O2_AV both in the Long-Run and/or in the Short_Run? We know from the (2.21) that, O1_AV is strictly affected by its history. Now we explore the other part of this question.

$$(O1_AV)_{t-1} - \alpha - b(O2_AV)_{t-1} = \hat{u}_{t-1}$$

From the Table A.11 we estimated (2.25) and determined the estimated values of coefficients as follows (indicated by ^):

$\omega = 0$; the constant of equation is not significant.

$$\hat{a}_1 = 1.851072 ; \hat{a}_2 = -1.918075 ; \hat{a}_3 = 1.518946 ; \hat{a}_4 = -0.833930 ; \hat{a}_5 = 0.245955$$

$$\hat{e}_1 = 0.157783 ; \hat{e}_2 = -0.581780 ; \hat{e}_3 = 0.837195 ; \hat{e}_4 = -0.725692 ; \hat{e}_5 = 0.331308$$

$$\hat{e}_6 = -0.090326$$

$\hat{r} = -0.040167$: that is signified by “ARTIK” in Table A.11.

$$\sum \hat{e}_i \Rightarrow \hat{e}_1 + \hat{e}_2 + \hat{e}_3 + \hat{e}_4 + \hat{e}_5 + \hat{e}_6 = -0,0715$$

As we can easily see, the coefficients $\sum \hat{e}_i$ shows us the Short – Run effect of O2_AV on O1_AV, and they are all statistically significant with % 95 level.

2. Does O1_AV affect O2_AV in the Long-Run and/or in the Short-Run?

$$\Delta(O2_AV)_t = \varphi + \sum_{i=1}^m g_i \Delta(O1_AV)_{t-i} + \sum_{j=1}^v f_j \Delta(O2_AV)_{t-j} + s \hat{u}_{t-1} \quad (2.26)$$

From the Table A.12, with the same argument we arrive that:

$\varphi = 0$; constant is not significant.

$\hat{g}_1 = 0.126706$; $\hat{g}_2 = -0.458759$; $\hat{g}_3 = 0.692304$; $\hat{g}_4 = -0.687259$; $g_5 = 0.389431$;
 $\hat{g}_6 = -0.113222$

$\hat{f}_1 = 1.932209$; $\hat{f}_2 = -2.175236$; $\hat{f}_3 = 1.853593$; $\hat{f}_4 = -1.098811$; $\hat{f}_5 = 0.279673$

$\hat{s} = 0.008196$: that is signified by “ARTIK” in Table A.12. However, this coefficient is not significant statistically so we have to accept that : $\hat{s} = 0$.

As we can easily see, the sum of coefficients $\sum \hat{g}_i$ shows us the Short-Run effect of O2_AV on O1_AV, and they are all statistically significant with % 95 level.

$$\sum \hat{g}_i \Rightarrow \hat{g}_1 + \hat{g}_2 + \hat{g}_3 + \hat{g}_4 + \hat{g}_5 + \hat{g}_6 = -0,05$$

Now we can write Vector Error Correction Mechanism (VECM) form of these relations:

$$\begin{bmatrix} \Delta(O1_AV)_t \\ \Delta(O2_AV)_t \end{bmatrix} = \begin{bmatrix} \Gamma_{11}(L) & \Gamma_{12}(L) \\ \Gamma_{21}(L) & \Gamma_{22}(L) \end{bmatrix} \begin{bmatrix} \Delta(O1_AV)_{t-1} \\ \Delta(O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} r \\ s \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 \end{bmatrix} \begin{bmatrix} (O1_AV)_{t-1} \\ (O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix} \quad (2.27)$$

As we know from the (2.22) that in the second part of the above equation we have:

$$\hat{u}_{t-1} = \begin{bmatrix} \beta_1 & \beta_2 \end{bmatrix} \begin{bmatrix} (O1_AV)_{t-1} \\ (O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix}$$

Causalities:

For the “causalities” we have to control the F-Statistics (Because we have two different type of explanatory variables in the regressions) obtained in the regression of the equations (2.25) and (2.26) and its results are given by Table A.11 and Table A.12 by their orders.

1. Instantaneous Causality or Short – Run Effects:

i.) $\Gamma_{12}(L) = e_1L + e_2L^2 + e_3L^3 + e_4L^4 + e_5L^5 + e_6L^6$: gives us the Short-Run Effect of region O2_AV on region O1_AV.

In the Table A.11 the value of F-statistic is 365.9451. This is very significant.

So we explicitly say that, Non-Stationary data generated by O2_AV instantly causes the generation of Non-Stationary data by O1_AV .

And as, $\hat{e}_1 + \hat{e}_2 + \hat{e}_3 + \hat{e}_4 + \hat{e}_5 + \hat{e}_6 = -0,0715$, we say that, if O2_AV increases by 1 (one) then O1_AV increases by -0,0715 in the Short –Run.

ii.) $\Gamma_{21}(L) = g_1L + g_2L^2 + g_3L^3 + g_4L^4 + g_5L^5 + g_6L^6$: gives us the Short-Run Effect of O1_AV on O2_AV.

In the Table A.12 the value of F-statistic is 345.6778. This is very significant.

So we explicitly say that, Non-Stationary data generated by region O1_AV instantly causes the generation of Non-Stationary data in region O2_AV .

And as, $\hat{g}_1 + \hat{g}_2 + \hat{g}_3 + \hat{g}_4 + \hat{g}_5 + \hat{g}_6 = -0,05$, we say that, if O1_AV increases by 1 (one) then O2_AV increases by -0,05 in the Short –Run.

2. Long –Run Effects:

i.) $\hat{r}$ the estimated value of r : gives the Long –Run effect of O2_AV on O1_AV.

$\hat{r} = -0.040167$: with significance level % 95 in Table A.11 $\hat{r}$ is named by “ARTIK” in the Table A.11

So we explicitly say that, Non-Stationary data generated by region O2_AV causes the generation of Non-Stationary data in region O1_AV in the Long-Run.

ii.) $\hat{s}$ the estimated value of s : gives the Long –Run effect of O1_AV on O2_AV.

$\hat{s} = 0$: with significance level % 95 in Table A.12 , $\hat{s}$ is named by “ARTIK” in the Table A.12.

So we explicitly say that, Non-Stationary data generated by region O1_AV DOES NOT cause the generation of Non-Stationary data in region O2_AV in the Long-Run.

3. CONCLUSION

3.1 For the whole range of recordings of Alzheimer and Epilepsy

When the whole recording data of 76000 samples are examined it is found that neither signal is Non-Stationary. That means that, even if there may exist certain perturbations within some intervals of this whole sample, all of these perturbations disappear and the data rests stationary in the long-run. This is valid for both Alzheimer disease and Epilepsy disease.

3.2. For small subintervals of Alzheimer data

Even if very small subintervals of Alzheimer data are tested (512 samples), there exists no non-stationarity in the data. This means that, Alzheimer data can be always characterized by stationary data generated by unobserved sources, and the Spatiotemporal Kalman Filtering explained in part 2, can be easily and plausibly applied to determine the exact location of unobserved sources (voxels) and imaging and Brain mapping is truly possible and convenient.

3.3. For small subintervals of Epilepsy data⁶

As we indicated, we analyzed and tested the recordings of 30 individuals who are under the suspect of having Epilepsy disease.

1. We find that the recordings of 30 individuals from this group contains some sample subintervals in which the observed data has Non – Stationary character. According to best of our knowledge, this means that: All of these individuals have Epilepsy Disease. The Non – Stationarity in their data shows us the temporary effects of Epilepsy.

2. As we examined the data of one individual taken from 19 sources or region separately, we found that in our every test interval, the Non – Stationarity exists within some of these 19 different signals. The number of brain regions that generates Non-

⁶ For regressions Eviews 5.00 Software is used.

Stationary data also varies from one individual to another individual. We think that the reason is clear and occurs from the physiological differences among individuals.

3. However, there is a common discovery for all Epilepsy - diseased people. The signal sources or regions that generate Non- Stationary data have co-movements with its neighbours because, the Non-Stationary data generated by them have Co-Integration relation. This charecteristic behaviour is observed in recordings of all individuals. This strictly shows us that: Epilepsy is generated in only one region or from only one signal source in Brain, and after, it is diffused to other regions within milliseconds.

4. As we tested the observed data :

$$y_t - y_{t-1} = \alpha + \beta t + (\rho - 1)y_{t-1} + \sum_{i=1}^p \gamma_i (y_{t-i} - y_{t-i+1}) + \varepsilon_t \quad \text{where} \quad \varepsilon_t \sim N(0, \sigma)$$

Physiologically this means that:

$$V_t - V_{t-1} = \alpha + \beta t + (\rho - 1)V_{t-1} + \sum_{i=1}^p \gamma_i (V_{t-i} - V_{t-i+1}) + \varepsilon_t \quad \text{where} \quad \varepsilon_t \sim N(0, \sigma)$$

Where V_t is the recorded or observed Voltage. We found out that during some small intervals, V_t is Non-Stationary. From the fundemental electric theory we know that

$V = I \cdot R$ (The multiplication of current and resistance). So we have to search the reasons of Non-Stationarity of observed voltage data in their components: unobserved current and/or unobserved resistance data. Briefly, as observed voltage data is Non-Stationary then from the part 4.1 by ignoring its history more than $p > 2$ without loss of any generality; we can define it as follows for theoretical discussion:

$$V_t = V_{t-1} + u_t \quad \text{where } u_t \text{ is AR}(p) \text{ noise or error or innovation or shock.}$$

Then we can write it in terms of current and resistance in three possible forms:

$$1.) (I.R)_t = (I.R)_{t-1} + u_t \Leftrightarrow (I_t.R_t) = (I_{t-1}.R_{t-1}) + u_t$$

$$\Leftrightarrow (I_t.R_{t-i}) = (I_{t-1}.R_{t-i-1}) + u_t; \quad \text{where } i \geq 1$$

$$2.) I.(R)_t = I.(R)_{t-1} + u_t$$

$$3.) (I)_t.R = (I)_{t-1}.R + u_t$$

Now, we have to explore the main reasons of Non-Stationary data generation by some regions in Brains of Epilepsy-diseased individuals among these 3 equations cited above.

We know that the resistance of voxels or neurons depends only their structural (biological) properties. However the quantity of current passing through them is not structural, instead depends on instantaneous events occurring within the cells and instantenous behaviours of ions.

Now we can argue the reasons of Epilepsy crise, its types and its clinical diagnosis types in the light of our findings:

Let's examine the first equation, it says that: the first part, and the second part is same $(I.R)_t = (I.R)_{t-1} + u_t \Leftrightarrow (I_t.R_t) = (I_{t-1}.R_{t-1}) + u_t$ and this indicates that, the current and the resistance that is subjected to the current change synchronizationally. We know from neurology that, the electrical structure of Brain cells can not give synchron response to the instantenous changes in current. If that was exist, neither crise can be temporary, hence the first or second sub-case is not possible. The third part of first equation claims a different finding: $\Leftrightarrow (I_t.R_{t-i}) = (I_{t-1}.R_{t-i-1}) + u_t$; where $i \geq 1$; that is, resistance of Brain cells give delayed response to the changes in current. This is physiologically and neurologically plausible. Note that, this case is possible.

Let's examine the second equation:

$I.(R)_t = I.(R)_{t-1} + u_t$; this Non –Stationary process shows us that although the current remain unchanged with time, the resistance of cells instantenously changes. Even if this could occur, we have to find a reason different from the electrical activity that changes the resistance of cells. To the best of our knowledge, there exists no neurological finding that claims electrical resistance of Brain cells can instantenously change independently from current such that observed voltage data becomes Non-Stationary. Even if this occurs, medical research must have reached clinical diagnosis (observation) of this case, because this case do cause always remaning and always varying (but not temporary) situations or crises or mental abnormalities. Finally the second case is not possible.

Let's examine the last equation: $(I)_t.R = (I)_{t-1}.R + u_t$; this process tells us that: when the resistance of cells remains unchanged, the current instantaneously changes in time such that Non-stationarity occurs in observed voltage data. This make sense, because we know that the variation of current is not structural (that is: not depends on biochemical properties of cells) but occurs instantaneously and disappears instantaneously. We think that most of the known – common Epilepsy crise types derives from this case.

Finally, from the argument above we obtained two main reasons that explain the nature and the types of Epilepsy crises and also we infer from them that two types of symptoms can be observed in Epilepsy-diseased individuals, these are:

$$1.) (I_t.R_{t-i}) = (I_{t-1}.R_{t-i-1}) + u_t ; \quad \text{where } i \geq 1 \quad (3.1)$$

$$2.) (I)_t.R = (I)_{t-1}.R + u_t \quad (3.2)$$

Comparison of Our Findings with Assumptions of Spatiotemporal Models in part 2 and Appendix of our thesis.

Now we have to indicate and focus on the “unplausible” strong assumptions of Spatiotemporal Models and Spatiotemporal Kalman filtering explained in parts 2 and 3 of our thesis.

Let's look at the (1.1) which is the assumed model of observed EEG according to these models. We renumber that equation (1.1) here as (3.3):

$$Y(t) = KJ(t) + \varepsilon(t) \quad \text{and where } \varepsilon(t) \sim N(0, \sigma) \quad (3.3)$$

As in the part 2 of our thesis, following from the literature, we know that in Spatiotemporal Models, this equation shows the link between observed EEG data $Y(t)$ and unobserved data generated by signal sources in Brain $X(t)$.

Now look at the state equation of this system (1.15) as shown in part 1. We renumber that equation (1.15) here as (3.4)

$$J(t) = AJ(t-1) + \eta(t) \quad (3.4)$$

and where the error is $\eta(t) \sim N(0, C_\eta)$ and where $C_\eta = E(\eta\eta^T)$

These equations (3.3) and (3.4) of Spatiotemporal Models together imply that:

$Y(t)$ is the observed Voltage data; $J(t)$ is unobserved Current data;

So we infer that K matrice in (3.3) shows the resistance data which links Voltage and Current in (3.3).

And the inventors of Spatiotemporal Models within last 5 years, assumed also that unobserved current is AR(1), as we can easily see from (3.4).

We will now compare these assumptions with our findins (3.1) and (3.2)

i) K in their models refers our R in (3.1) and (3.2). They assume K as a constant parameter. Even if we accept that resistance K (in our findings R) to be constant like in our (3.2), we can not accept that unobserved Current $J(t)$ (in our Equations I_t) is AR(1).

ii.) The Spatiotemporal models does not assume or estimate any equation like our finding (3.1)

Our critics are also valid for the Spatiotemporal system constructed with (3.18) and (3.24) in part 3 as follows: We renumber the equations (3.18) and (3.24) here as (3.5) and (3.6) respectively:

$$Y(k) = KJ(k) + \varepsilon(k) \quad \text{and where} \quad \varepsilon(k) \sim N(0, \tau) \quad (3.5)$$

$$J(k) = A_1 J(k-1) + A_2 J(k-2) + H(k) \quad \text{and where} \quad H(k) \sim N(0, C_\eta) \quad \text{and where}$$

$$C_\eta = \sigma_\eta^2 (L_{N_j}^T L_{N_j})^{-1} \quad (3.6)$$

These comparisons show us that, the newest imaging techniques and Brain Mapping techniques, can capture only small amount of the “disordered” or Epilepsy signal sources that is shown by (3.2). Moreover, they never can image the signal sources which behave like (3.1). Finally, all the imaging techniques are insufficient to capture or image the second type of Epilepsy (3.1), and are capable to image only small amount of its first

type (3.2). Hence, these Spatiotemporal models underestimate or misestimate the epileptic signal sources in the brain.

3.3.1 Auxiliary Questions and Discussion

Now we try to find answers of other questions than given in Introduction and which can be derived from the findings cited above.

If there exist two main reasons of Epilepsy crises indicated above with (3.1) and (3.2) and the second one (3.2) : $(I)_t.R = (I)_{t-1}.R + u_t$ causes most of the common Epilepsy crises as we claimed, can we explain how an Epilepsy crise starts? Because as we know, in “normal” human brain the current also is not constant in time. What difference occurs in the Brain of Epilepsy-diseased individual that the sources or regions begin to generate, at any instant of time, Non-Stationary data? And from which reasons this Non-Stationary data becomes Stationary “shortly”? What symptoms can be “observed”? In fact these symptoms are similiar or completely different from each other? What are the main differences between these two types Epilepsy crises?

We think that even if there may be several reasons, these reasons all originate from the difference structural properties of resistance of our brains’ cells (shown with R in the (3.2)) and so the very similiar symptoms can be detected: First, the structural resistance of Brain cells (their electrical properties) of Epilepsy-diseased individuals may have “*narrow electrical thresholds*” when compared to the that of “normal” brain’s cells. Second reason may be biochemical: the conductivity of Brain cell liquid of Epilepsy-diseased individuals may be very high when compared to the that of “normal” brains. The symptoms of this case must be “observedly - measuredly” same: these types of crises diffuse very fast and lose their effects very fast. So we can give the answer of last question cited above: inhibitors in the brain cells terminate the crises and as the brain cells are not structurally pertubated yet (R remains unchanged in the (3.2)), the crise or the shocks can be absorbed easily and hence we observe that the behavior of data transforms from Non-stationary to Stationary.

We ask the same questions for our other finding given with (3.1):

$$(I_t.R_{t-i}) = (I_{t-1}.R_{t-i-1}) + u_t ; \quad \text{where } i \geq 1$$

Although the definition of current in this equation is same with the equation analyzed above, the symptoms of this case must be completely different: First, the equation tells us that, the changes in current are such that both the data generated by sources or regions (cells or voxels) become Non-Stationary and the resistance of the brain cells that are subjected to this event perturbates within time (lagged) but not at the same time.

This case causes intense crises. By the way, even if the effects of these type of crises also like other types cited above temporary, the duration of crise must be longer. The main difference between these type of crises and others explained above is: The second one can or can not capture all regions as fast as other types, but as it perturbates the structure of cells by its higher impact, these second type crises increases potential duration of possible crises in future by decreasing this possibility of new great crises in future. This result is very interesting. The main reason of this “imaginary anthogonism” is that: the perturbation of resistance of brain cells of Epilepsy-diseased individuals does not cause totally vanishing of these cells, so they can still absorb the spikes and random shocks but luckily can not propogates them quickly.

Triggering Of Non-Stationary Data Generation in one region by another region of a Brain of Epilepsy-Diseased individual: shown by VECM :

$$\begin{bmatrix} \Delta(O1_AV)_t \\ \Delta(O2_AV)_t \end{bmatrix} = \begin{bmatrix} \Gamma_{11}(L) & \Gamma_{12}(L) \\ \Gamma_{21}(L) & \Gamma_{22}(L) \end{bmatrix} \begin{bmatrix} \Delta(O1_AV)_{t-1} \\ \Delta(O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} r \\ s \end{bmatrix} \begin{bmatrix} \beta_1 & \beta_2 \end{bmatrix} \begin{bmatrix} (O1_AV)_{t-1} \\ (O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix}$$

Here technically we are also capable to determine the exact number of the dependence of the signal values to its own history.

In our experiments over an individual; the triggering relation between O1_AV and O2_AV is shown by its numerical estimated values:

$$\Gamma_{11}(L) = a_1L + a_2L^2 + a_3L^3 + a_4L^4 + a_5L^5$$

$$\Gamma_{12}(L) = e_1L + e_2L^2 + e_3L^3 + e_4L^4 + e_5L^5 + e_6L^6$$

$$\Gamma_{21}(L) = g_1L + g_2L^2 + g_3L^3 + g_4L^4 + g_5L^5 + g_6L^6$$

$$\Gamma_{22}(L) = f_1L + f_2L^2 + f_3L^3 + f_4L^4 + f_5L^5$$

With their estimated values:

$$1. \hat{a}_1 = 1.851072 ; \hat{a}_2 = -1.918075 ; \hat{a}_3 = 1.518946 ; \hat{a}_4 = -0.833930 ; \hat{a}_5 = 0.245955$$

$$2. \hat{e}_1 = 0.157783 ; \hat{e}_2 = -0.581780 ; \hat{e}_3 = 0.837195 ; \hat{e}_4 = -0.725692 ; \hat{e}_5 = 0.331308 ; \hat{e}_6 = -0.090326$$

$$3. \hat{g}_1 = 0.126706 ; \hat{g}_2 = -0.458759 ; \hat{g}_3 = 0.692304 ; \hat{g}_4 = -0.687259 ; \hat{g}_5 = 0.389431 ; \hat{g}_6 = -0.113222$$

$$4. \hat{f}_1 = 1.932209 ; \hat{f}_2 = -2.175236 ; \hat{f}_3 = 1.853593 ; \hat{f}_4 = -1.098811 ; \hat{f}_5 = 0.279673$$

In our statistical analysis example for an epileptic patient; the triggering relation between O1_AV and O2_AV is shown by its numerical estimated values:

$$\begin{bmatrix} \Delta(O1_AV)_t \\ \Delta(O2_AV)_t \end{bmatrix} = \begin{bmatrix} 0,8639 & -0,0715 \\ -0,051 & 0,7914 \end{bmatrix} \begin{bmatrix} \Delta(O1_AV)_{t-1} \\ \Delta(O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} -0,04 \\ 0 \end{bmatrix} \begin{bmatrix} 1 & -0,57 \end{bmatrix} \begin{bmatrix} (O1_AV)_{t-1} \\ (O2_AV)_{t-1} \end{bmatrix} + \begin{bmatrix} \varepsilon_{1t} \\ \varepsilon_{2t} \end{bmatrix}$$

Clearly we can estimate the region which creates Non-Stationary Data in Brain of Epilepsy-diseased individual both in Short-Run and Long-Run.

As an extension we propose to make a study to determine exact center of Epilepsy by the method of Panel Co-Integration. That will explicitly give us, which region of the 19 regions in the brain creates the Crises.

FIGURES

In all Figures below except Figure A.15,A.16,A.17 the y-axis indicates measured EEG value, while x-axis indicates the measurement order or sample number.

Figures A.15,A.16 and A.17 are taken for showing the similarity of datas examined in Economics and EEG data. These figures show certain economical variables changing with time.

EXAMPLES OF MEASUREMENTS FROM INDIVIDUAL No:1 UNDER THE SUSPECT OF EPILEPSY

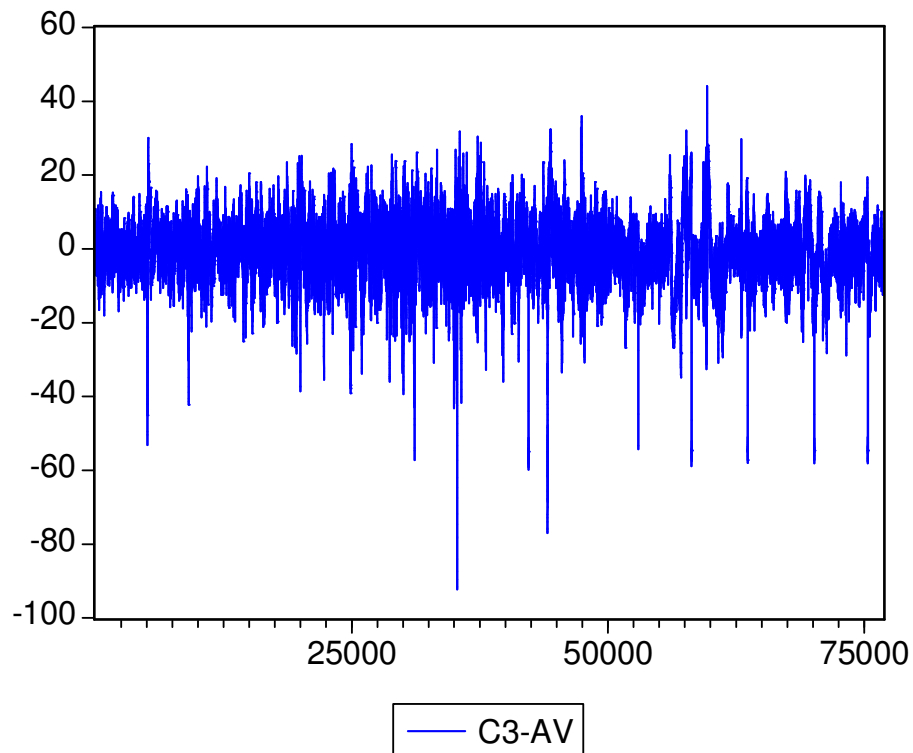


Figure A.1: 76000 Measurements from C3_AV channel

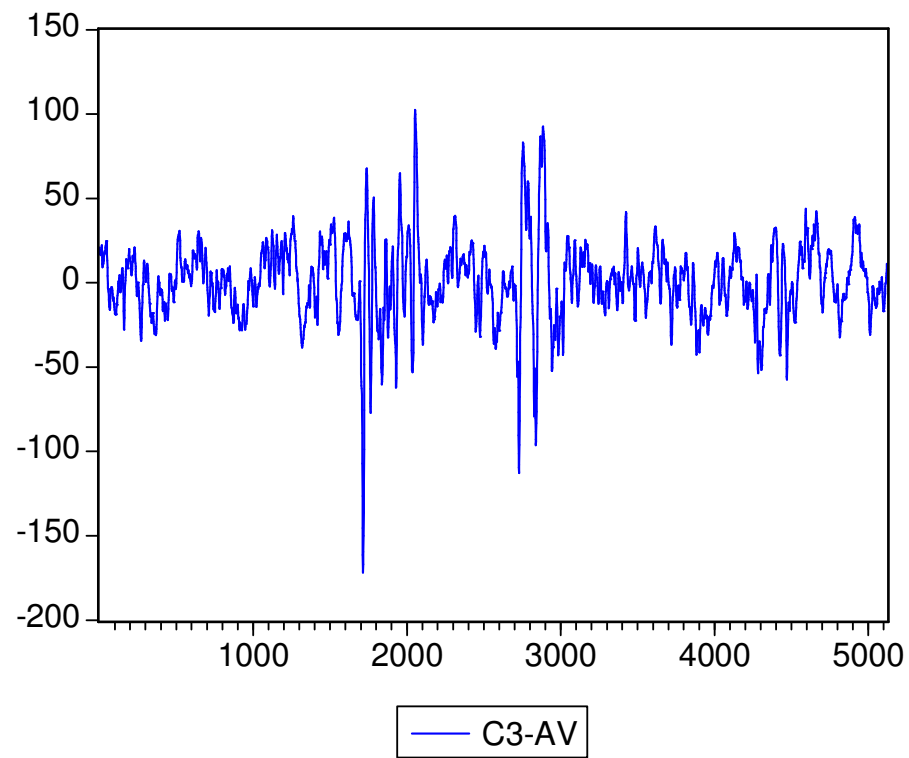


Figure A.2: 5000 Measurements from C3_AV channel

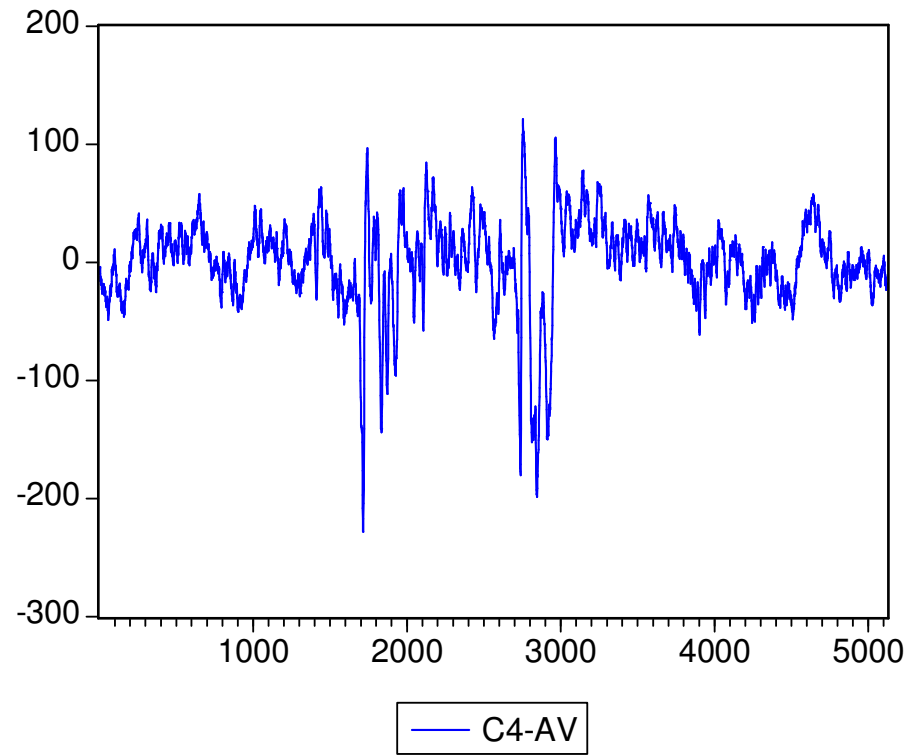


Figure A.3: 5000 Measurements from C4_AV channel

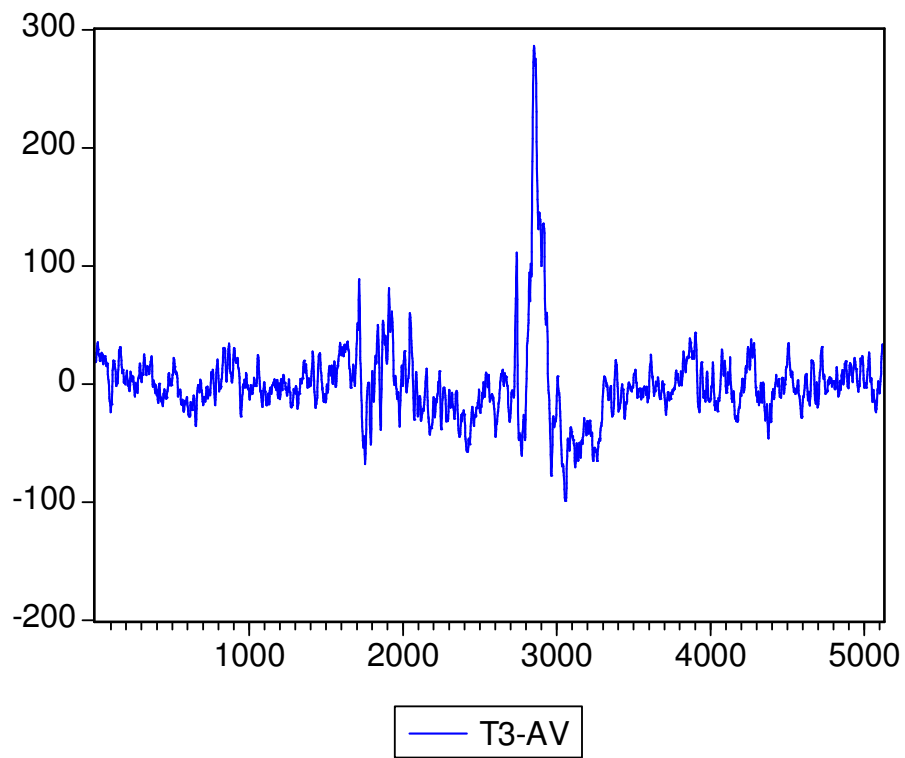


Figure A.4: 5000 Measurements from T3_AV channel

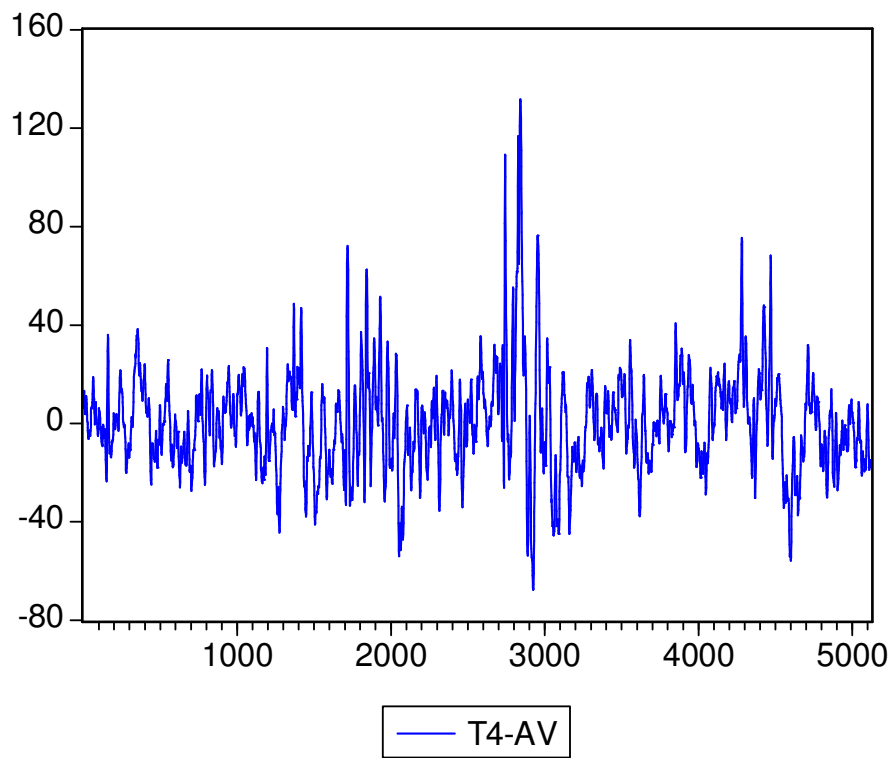


Figure A.5: 5000 Measurements from T4_AV channel

**EXAMPLES OF MEASUREMENTS FROM INDIVIDUAL No:2 UNDER THE
SUSPECT OF EPILEPSY**

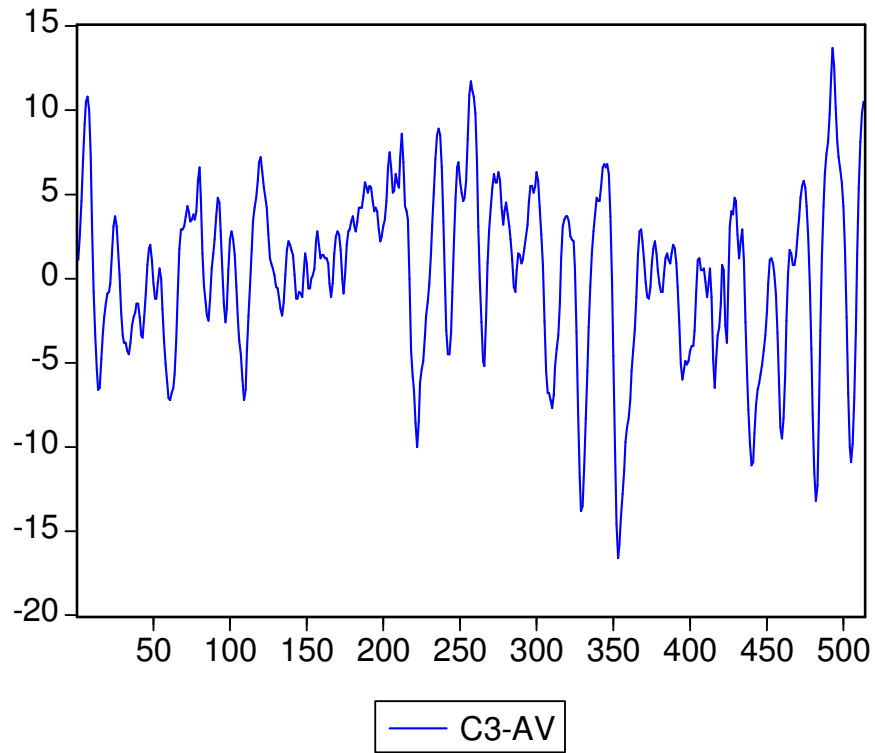


Figure A.6: 500 Measurements from C3_AV channel

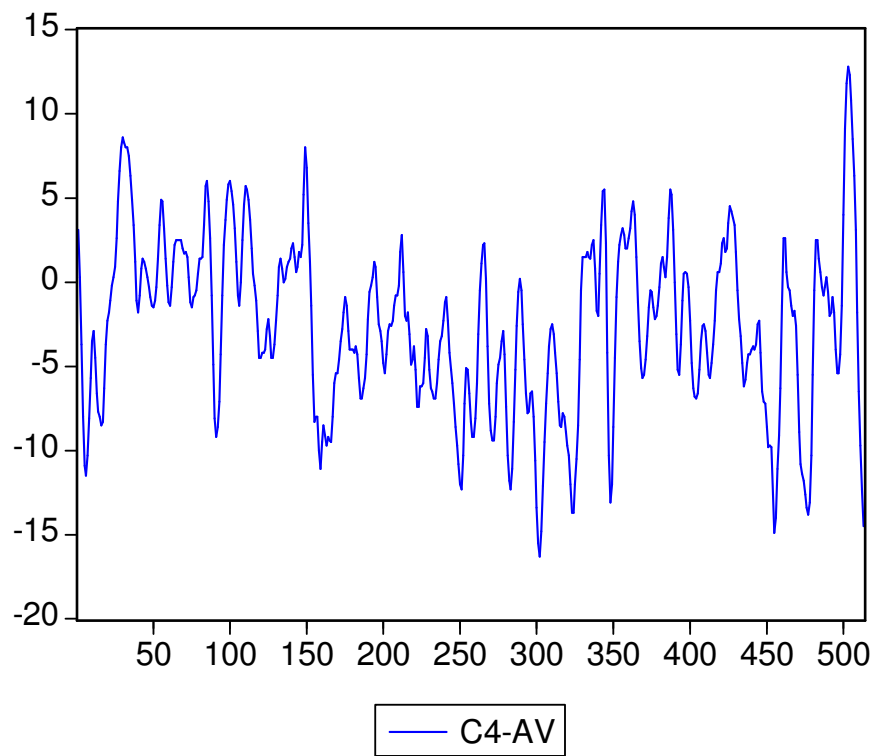


Figure A.7: 500 Measurements from C4_AV channel

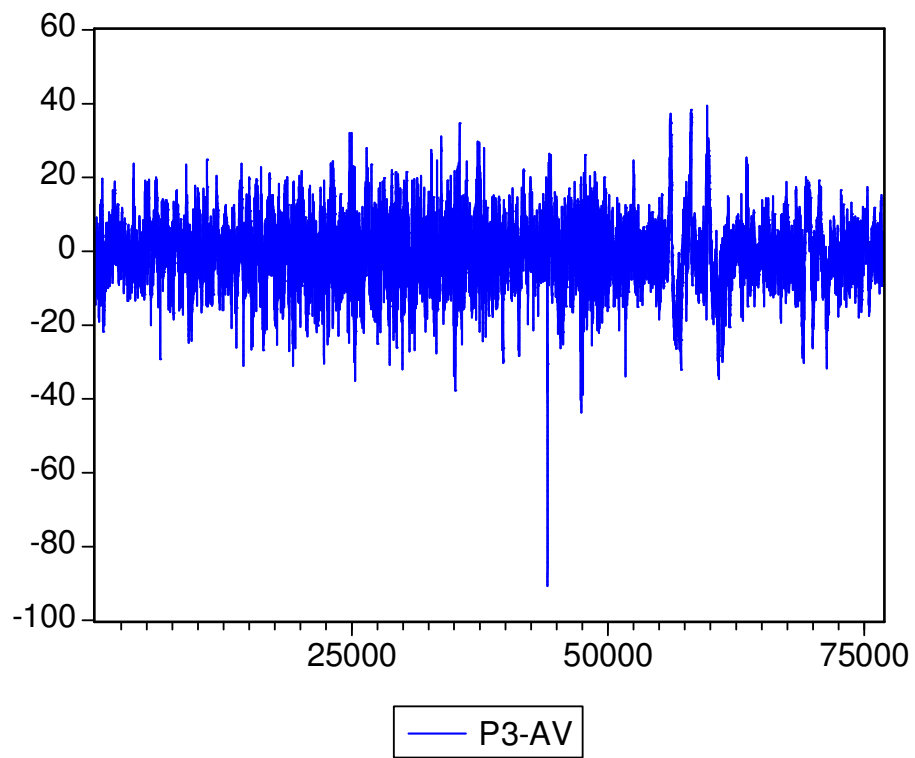


Figure A.8: 76000 Measurements from P3_AV channel

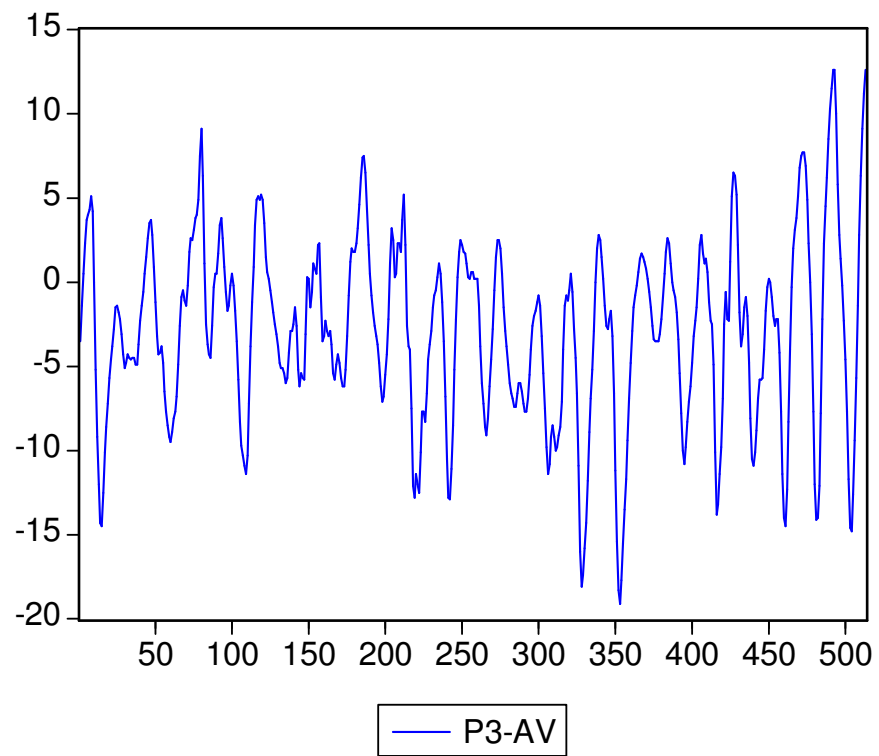


Figure A.9: 500 Measurements from P3_AV channel

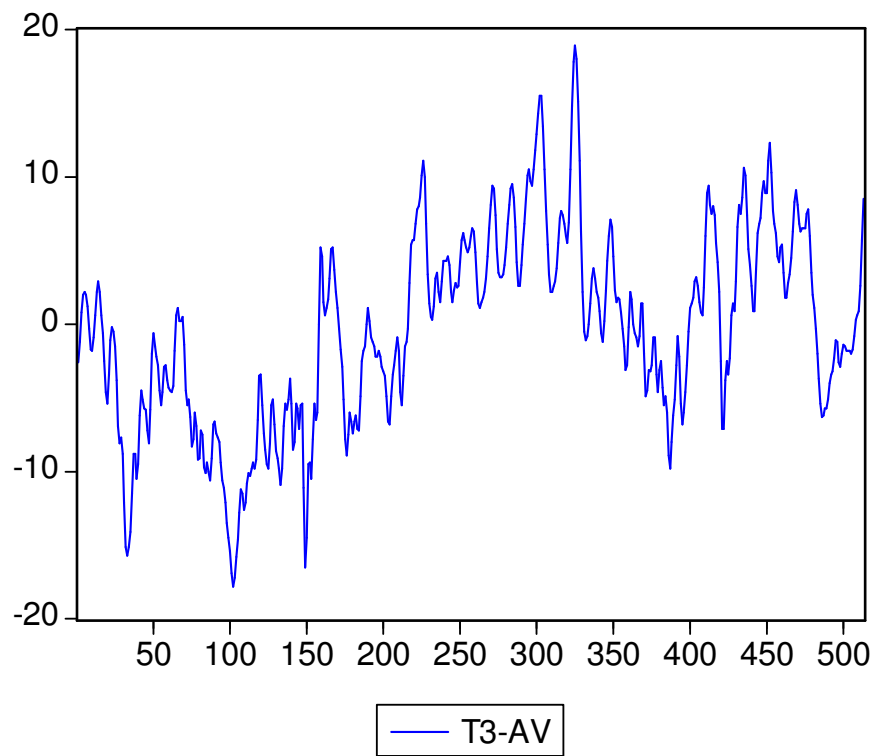


Figure A.10: 500 Measurements from T3_AV channel

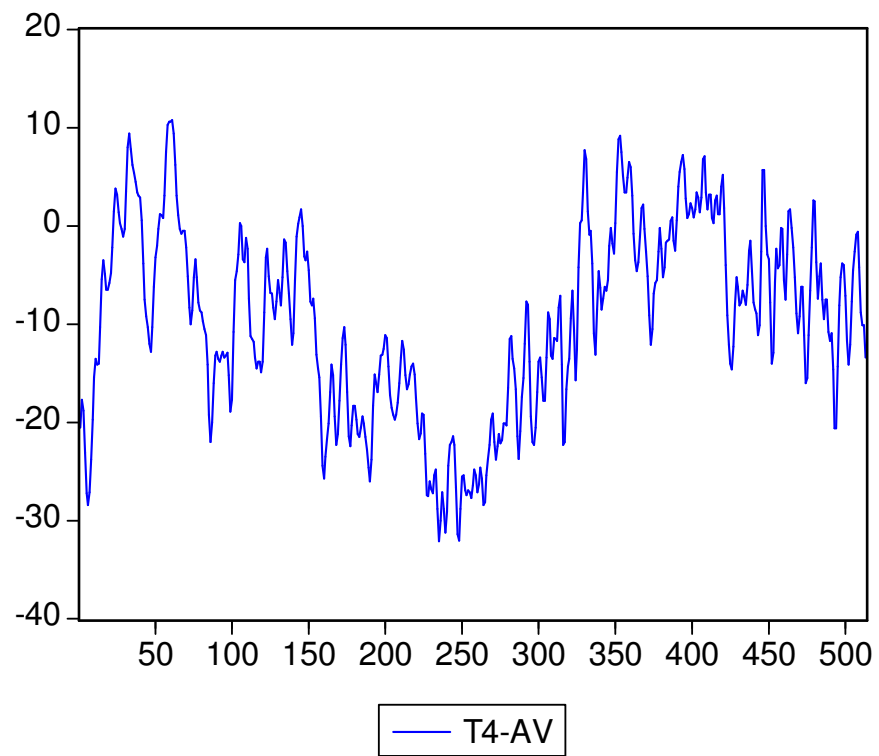


Figure A.11: 500 Measurements from T4_AV channel

**EXAMPLES OF MEASUREMENTS FROM INDIVIDUAL No:3 UNDER THE
SUSPECT OF EPILEPSY**

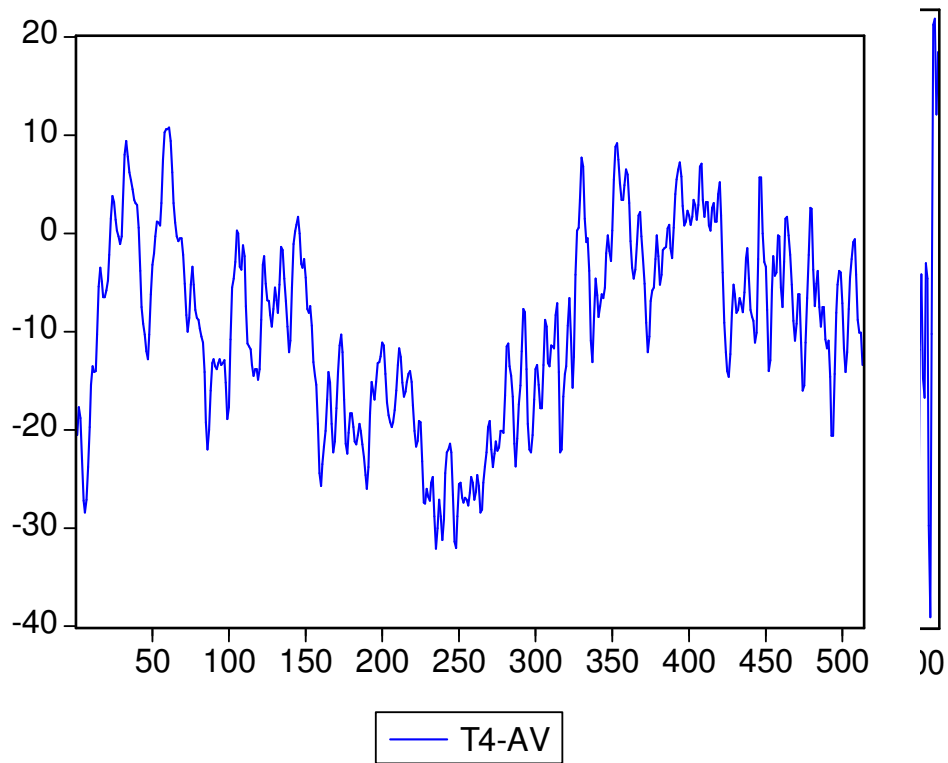


Figure A.12: 500 Measurements from C4_AV channel

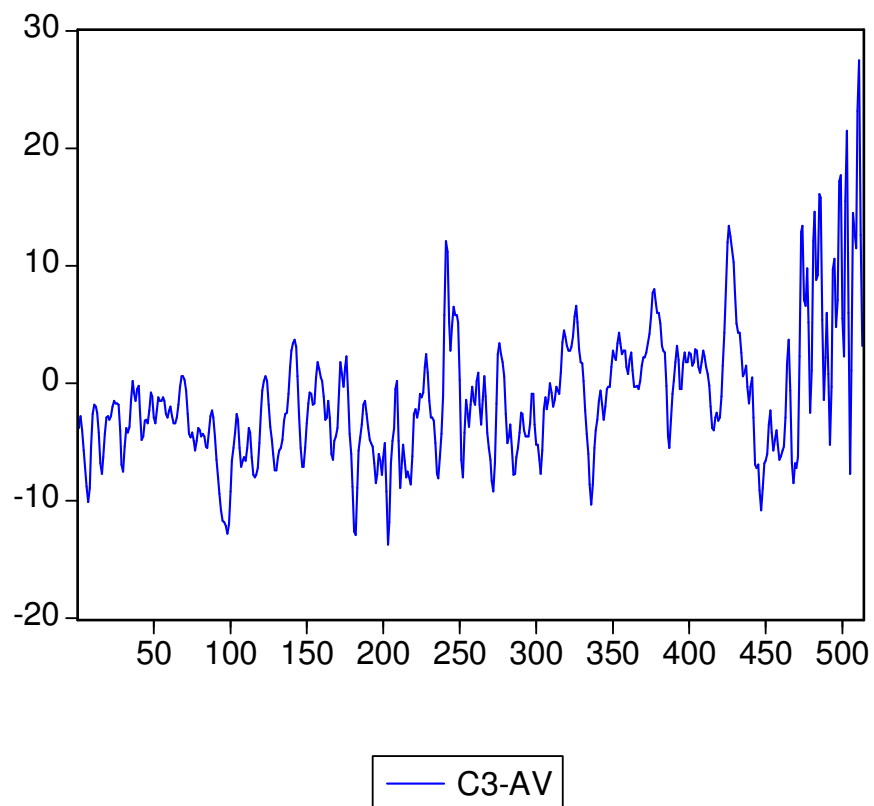


Figure A.13: 500 Measurements from C3_AV channel

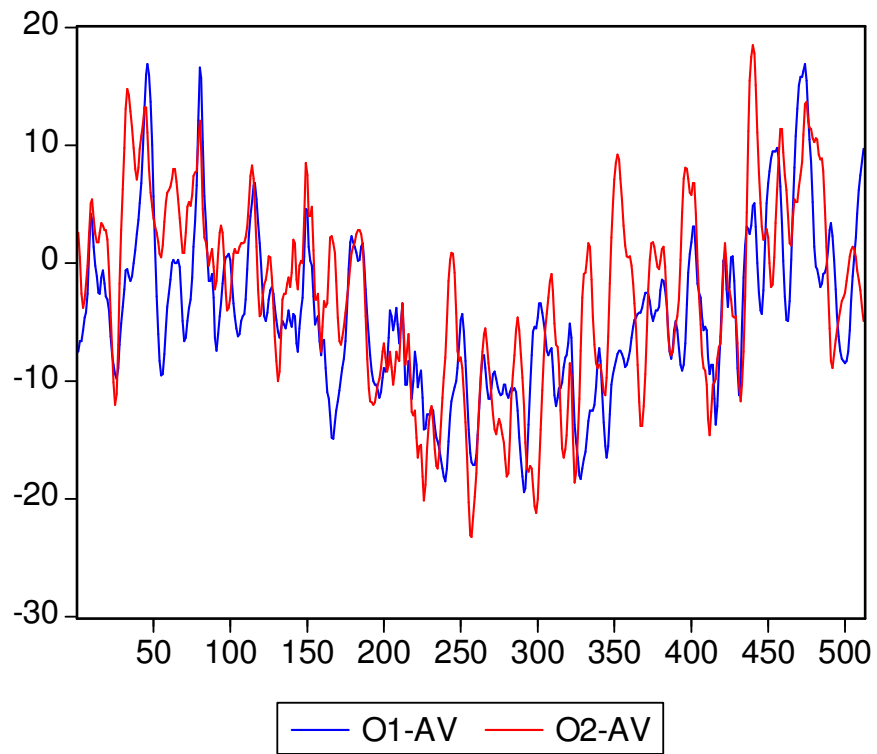


Figure A.14: Co – Movement of O1-AV and O2-AV

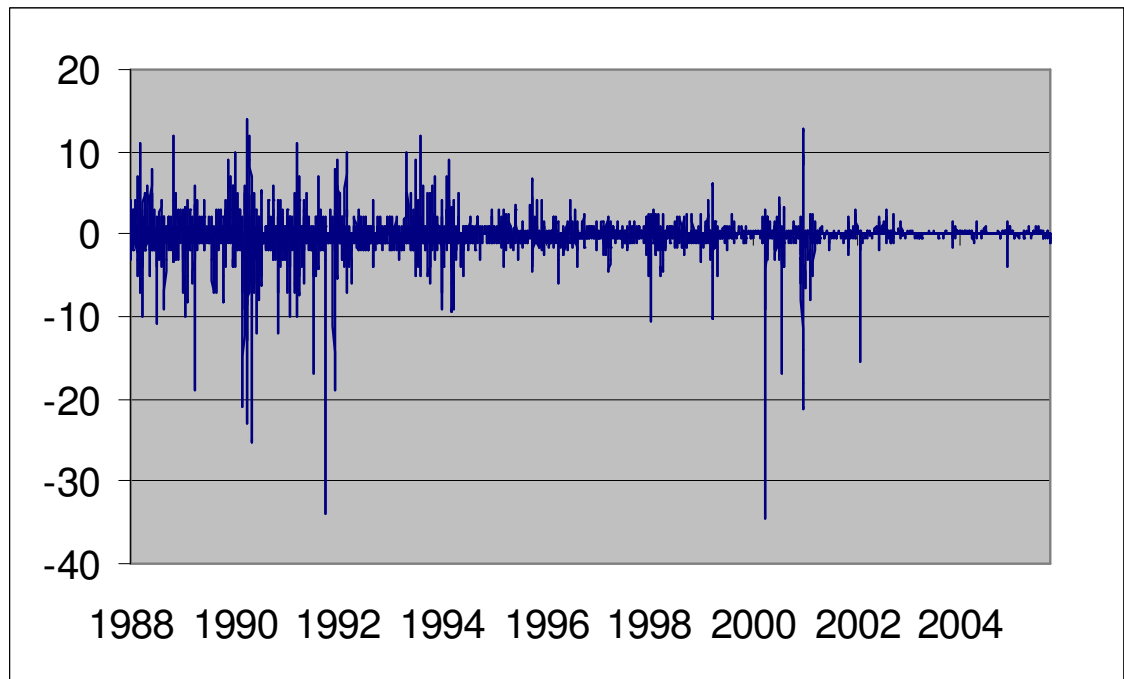


Figure A.15: The change on day d of contract for the current month of USA Central Bank funds

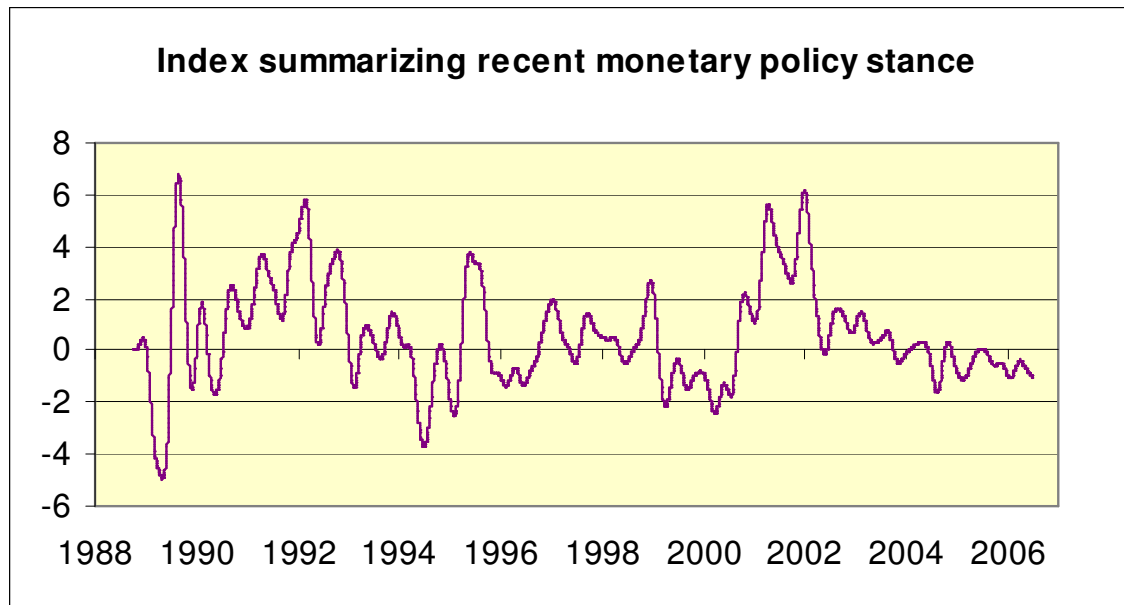


Figure A.16

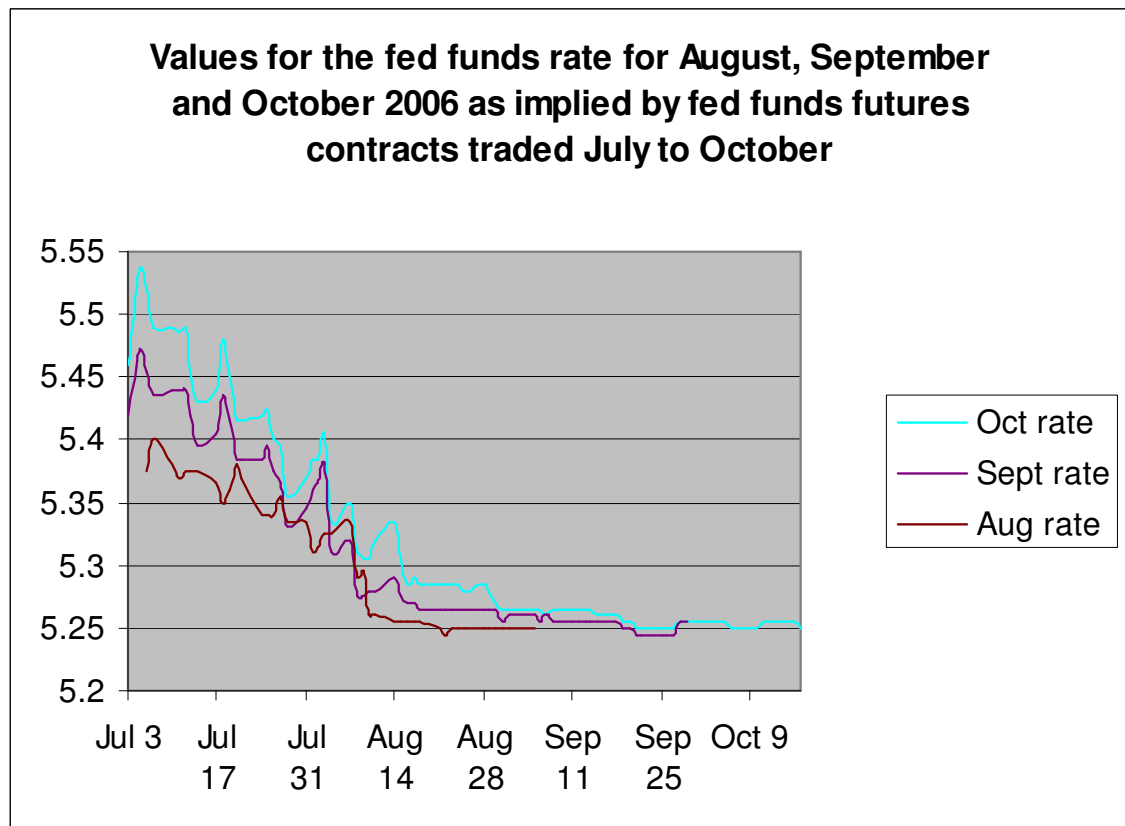


Figure A.17

Table A.1: Regression of first difference of EEG data taken from T4_AV Ch. For 512 Samples

Dependent Variable: D(T4_AV)

Method: Least Squares

Date: 09/05/06 Time: 22:32

Sample (adjusted): 11 513

Included observations: 503 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
T4_AV(-1)	-0.004617	0.002689	-1.717265	0.0866
D(T4_AV(-1))	2.084047	0.042991	48.47644	0.0000
D(T4_AV(-2))	-2.830961	0.098159	-28.84049	0.0000
D(T4_AV(-3))	2.487228	0.151528	16.41431	0.0000
D(T4_AV(-4))	-1.233132	0.179296	-6.877627	0.0000
D(T4_AV(-5))	-0.377646	0.186062	-2.029671	0.0429
D(T4_AV(-6))	1.198170	0.178901	6.697373	0.0000
D(T4_AV(-7))	-1.132620	0.151455	-7.478251	0.0000
D(T4_AV(-8))	0.678009	0.097986	6.919443	0.0000
D(T4_AV(-9))	-0.273772	0.043141	-6.346010	0.0000
R-squared	0.919102	Mean dependent var		0.003976
Adjusted R-squared	0.917626	S.D. dependent var		2.682484
S.E. of regression	0.769898	Akaike info criterion		2.334563
Sum squared resid	292.2223	Schwarz criterion		2.418472
Log likelihood	-577.1427	Durbin-Watson stat	1.976500	

Table A.4: Regression of first difference of EEG data taken from T3_AV Ch. For 512 Samples

Dependent Variable: D(T3_AV)

Method: Least Squares

Date: 09/05/06 Time: 22:57

Sample (adjusted): 16 513

Included observations: 498 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
T3_AV(-1)	-0.007879	0.004107	-1.918365	0.0557
D(T3_AV(-1))	1.786066	0.043046	41.49200	0.0000
D(T3_AV(-2))	-1.833752	0.090612	-20.23737	0.0000
D(T3_AV(-3))	1.047938	0.123872	8.459815	0.0000
D(T3_AV(-4))	0.077135	0.131632	0.585991	0.5582
D(T3_AV(-5))	-0.897817	0.126938	-7.072900	0.0000
D(T3_AV(-6))	0.526754	0.131099	4.017996	0.0001
D(T3_AV(-7))	0.418830	0.134194	3.121067	0.0019
D(T3_AV(-8))	-0.818682	0.127464	-6.422850	0.0000
D(T3_AV(-9))	0.451733	0.132238	3.416047	0.0007
D(T3_AV(-10))	-0.039466	0.130960	-0.301361	0.7633
D(T3_AV(-11))	-0.277006	0.102987	-2.689727	0.0074
D(T3_AV(-12))	0.260747	0.051799	5.033781	0.0000
D(T3_AV(-14))	-0.152238	0.021082	-7.221108	0.0000
R-squared	0.899830	Mean dependent var		0.012651
Adjusted R-squared	0.897139	S.D. dependent var		1.614902
S.E. of regression	0.517930	Akaike info criterion		1.549755
Sum squared resid	129.8336	Schwarz criterion		1.668126
Log likelihood	-371.8891	Durbin-Watson stat		1.974601

Table A.5: Regression of first difference of EEG data taken from T3_AV Ch.
For 2048 samples.

Dependent Variable: D(T3_AV)

Method: Least Squares

Date: 09/05/06 Time: 23:11

Sample (adjusted): 18 2049

Included observations: 2032 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
T3_AV(-1)	-0.008060	0.001805	-4.465237	0.0000
D(T3_AV(-1))	1.766175	0.022120	79.84475	0.0000
D(T3_AV(-2))	-1.709735	0.045109	-37.90263	0.0000
D(T3_AV(-3))	0.918891	0.058900	15.60099	0.0000
D(T3_AV(-4))	0.141761	0.062291	2.275780	0.0230
D(T3_AV(-5))	-0.845738	0.062299	-13.57557	0.0000
D(T3_AV(-6))	0.476706	0.064752	7.361973	0.0000
D(T3_AV(-7))	0.333313	0.065495	5.089154	0.0000
D(T3_AV(-8))	-0.537462	0.065889	-8.157138	0.0000
D(T3_AV(-9))	0.096606	0.065879	1.466420	0.1427
D(T3_AV(-10))	0.182190	0.065533	2.780127	0.0055
D(T3_AV(-11))	-0.286849	0.064843	-4.423760	0.0000
D(T3_AV(-12))	0.139873	0.062345	2.243541	0.0250
D(T3_AV(-13))	0.127775	0.062361	2.048945	0.0406
D(T3_AV(-14))	-0.170433	0.059031	-2.887186	0.0039
D(T3_AV(-15))	-0.059148	0.045052	-1.312879	0.1894
D(T3_AV(-16))	0.079939	0.022233	3.595561	0.0003
R-squared	0.888579	Mean dependent var		0.001969

Adjusted R-squared	0.887694	S.D. dependent var	1.461697
S.E. of regression	0.489845	Akaike info criterion	1.418874
Sum squared resid	483.4949	Schwarz criterion	1.465865
Log likelihood	-1424.576	Durbin-Watson stat	1.991163

Table A.6: Non-Stationary Test for EEG data taken from O1_AV Ch. For 512 Samples
for 0,025 : I(1)

Dependent Variable: D(O1_AV)

Method: Least Squares

Date: 09/06/06 Time: 00:19

Sample (adjusted): 18 513

Included observations: 496 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
O1_AV(-1)	-0.006859	0.003128	-2.192682	0.0288
D(O1_AV(-1))	1.851927	0.045180	40.98953	0.0000
D(O1_AV(-2))	-1.797980	0.095831	-18.76206	0.0000
D(O1_AV(-3))	1.041439	0.125542	8.295523	0.0000
D(O1_AV(-4))	-0.037885	0.133473	-0.283840	0.7767
D(O1_AV(-5))	-0.683520	0.132944	-5.141407	0.0000
D(O1_AV(-6))	0.300666	0.135993	2.210893	0.0275
D(O1_AV(-7))	0.577821	0.136597	4.230108	0.0000
D(O1_AV(-8))	-0.788105	0.138689	-5.682551	0.0000
D(O1_AV(-9))	0.268641	0.138955	1.933305	0.0538
D(O1_AV(-10))	0.047404	0.137445	0.344898	0.7303
D(O1_AV(-11))	-0.191605	0.136318	-1.405570	0.1605
D(O1_AV(-12))	0.090942	0.132372	0.687024	0.4924
D(O1_AV(-13))	0.201007	0.132664	1.515157	0.1304
D(O1_AV(-14))	-0.219053	0.124900	-1.753833	0.0801
D(O1_AV(-15))	-0.064491	0.094946	-0.679243	0.4973
D(O1_AV(-16))	0.121471	0.045104	2.693117	0.0073
R-squared	0.918453	Mean dependent var		0.021573

Adjusted R-squared	0.915729	S.D. dependent var	1.699661
S.E. of regression	0.493402	Akaike info criterion	1.458687
Sum squared resid	116.6102	Schwarz criterion	1.602864
Log likelihood	-344.7543	Durbin-Watson stat	1.985922

Table A.7: Non-Stationary Test for EEG data taken from O2_AV Ch. For 512 Sample

Dependent Variable: D(O2_AV)

Method: Least Squares

Date: 09/05/06 Time: 23:26

Sample (adjusted): 15 480

Included observations: 466 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
O2_AV(-1)	-0.006196	0.003726	-1.662994	0.0970
D(O2_AV(-1))	1.957659	0.044298	44.19309	0.0000
D(O2_AV(-2))	-2.126221	0.098100	-21.67392	0.0000
D(O2_AV(-3))	1.447384	0.137204	10.54914	0.0000
D(O2_AV(-4))	-0.289979	0.152754	-1.898337	0.0583
D(O2_AV(-5))	-0.806779	0.153678	-5.249792	0.0000
D(O2_AV(-6))	0.822056	0.157728	5.211847	0.0000
D(O2_AV(-7))	-0.110391	0.162216	-0.680521	0.4965
D(O2_AV(-8))	-0.355134	0.157360	-2.256830	0.0245
D(O2_AV(-9))	0.092161	0.153471	0.600510	0.5485
D(O2_AV(-10))	0.251595	0.153243	1.641799	0.1013
D(O2_AV(-11))	-0.567131	0.138228	-4.102853	0.0000
D(O2_AV(-12))	0.592789	0.098480	6.019358	0.0000
D(O2_AV(-13))	-0.311221	0.044789	-6.948543	0.0000
R-squared	0.922652	Mean dependent var	0.018240	
Adjusted R-squared	0.920427	S.D. dependent var	2.006610	
S.E. of regression	0.566038	Akaike info criterion	1.729271	
Sum squared resid	144.8203	Schwarz criterion	1.853774	
Log likelihood	-388.9201	Durbin-Watson stat	2.170445	

Table A.8: Co-Integration Test-1 for EEG datas taken from O1_AV and O2_AV

Date: 09/06/06 Time: 00:40

Sample (adjusted): 6 513

Included observations: 508 after adjustments

Trend assumption: No deterministic trend

Series: O1_AV O2_AV

Lags interval (in first differences): 1 to 4

Unrestricted Cointegration Rank Test (Trace)

Hypothesized		Trace 0.05		
No. of CE(s)	Eigenvalue	Statistic	Critical Value	Prob.**
None *	0.081349	48.90161	12.32090	0.0000
At most 1 *	0.011349	5.798290	4.129906	0.0191

Trace test indicates 2 cointegrating eqn(s) at the 0.05 level

* denotes rejection of the hypothesis at the 0.05 level

**MacKinnon-Haug-Michelis (1999) p-values

Unrestricted Cointegration Rank Test (Maximum Eigenvalue)

Hypothesized		Max-Eigen 0.05		
No. of CE(s)	Eigenvalue	Statistic	Critical Value	Prob.**
None *	0.081349	43.10332	11.22480	0.0000
At most 1 *	0.011349	5.798290	4.129906	0.0191

Max-eigenvalue test indicates 2 cointegrating eqn(s) at the 0.05 level

* denotes rejection of the hypothesis at the 0.05 level

**MacKinnon-Haug-Michelis (1999) p-values

Unrestricted Cointegrating Coefficients (normalized by b'*S11*b=I):

O1_AV	O2_AV
-0.198467	0.202073
0.074328	0.065194

Unrestricted Adjustment Coefficients (alpha):

D(O1_AV)	0.119443	-0.046788
D(O2_AV)	-0.063243	-0.069936

1 Cointegrating Equation(s): Log likelihood -918.1643

Normalized cointegrating coefficients (standard error in parentheses)

O1_AV	O2_AV
1.000000	-1.018167
(0.10000)	

Adjustment coefficients (standard error in parentheses)

D(O1_AV)	-0.023706
(0.00529)	

D(O2_AV)	0.012552
(0.00613)	

Table A.9: Co-Integration Vector Test for O1_AV and O2_AV

Dependent Variable: O1_AV

Method: Least Squares

Date: 11/27/06 Time: 01:55

Sample: 1 512

Included observations: 512

Variable	Coefficient	Std. Error	t-Statistic	Prob.
O2_AV	0.570657	0.028419	20.08015	0.0000
C	-3.052537	0.241911	-12.61845	0.0000
R-squared	0.441532	Mean dependent var	-4.474609	
Adjusted R-squared	0.440437	S.D. dependent var	6.996951	
S.E. of regression	5.233996	Akaike info criterion	6.152126	
Sum squared resid	13971.30	Schwarz criterion	6.168682	
Log likelihood	-1572.944	F-statistic	403.2123	
Durbin-Watson stat	0.090684	Prob(F-statistic)	0.000000	

Table A.10: Non-Stationary Test for Residuals of Co-Integration Relation between O1_AV and O2_AV

Dependent Variable: D(CO)

Method: Least Squares

Date: 11/27/06 Time: 02:44

Sample (adjusted): 10 512

Included observations: 503 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
C	0.005134	0.020795	0.246882	0.8051
CO(-1)	-0.026357	0.006520	-4.042340	0.0001
D(CO(-1))	1.686711	0.040888	41.25239	0.0000
D(CO(-2))	-1.327579	0.085072	-15.60533	0.0000
D(CO(-3))	0.495686	0.103706	4.779723	0.0000
D(CO(-4))	0.248978	0.102422	2.430896	0.0154
D(CO(-5))	-0.567359	0.102411	-5.540023	0.0000
D(CO(-6))	0.204454	0.103461	1.976148	0.0487
D(CO(-7))	0.346806	0.084114	4.123036	0.0000
D(CO(-8))	-0.351022	0.042216	-8.314904	0.0000
R-squared	0.914183	Mean dependent var	0.022279	
Adjusted R-squared	0.912616	S.D. dependent var	1.576963	
S.E. of regression	0.466162	Akaike info criterion	1.331114	
Sum squared resid	107.1324	Schwarz criterion	1.415022	
Log likelihood	-324.7751	F-statistic	583.5301	
Durbin-Watson stat	1.960929	Prob(F-statistic)	0.000000	

Table A.11: VECM Test for first difference of EEG Data taken from O1_AV Ch.

For 512 Samples

Dependent Variable: D(O1_AV)

Method: Least Squares

Date: 11/27/06 Time: 19:50

Sample (adjusted): 8 512

Included observations: 505 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
C	0.001046	0.024393	0.042875	0.9658
D(O1_AV(-1))	1.851072	0.046726	39.61504	0.0000
D(O1_AV(-2))	-1.918075	0.093128	-20.59606	0.0000
D(O1_AV(-3))	1.518946	0.109252	13.90315	0.0000
D(O1_AV(-4))	-0.833930	0.089756	-9.291095	0.0000
D(O1_AV(-5))	0.245955	0.046388	5.302079	0.0000
D(O2_AV(-1))	0.157783	0.040707	3.876068	0.0001
D(O2_AV(-2))	-0.581780	0.086039	-6.761807	0.0000
D(O2_AV(-3))	0.837195	0.112152	7.464856	0.0000
D(O2_AV(-4))	-0.725692	0.113079	-6.417564	0.0000
D(O2_AV(-5))	0.331308	0.085242	3.886689	0.0001
D(O2_AV(-6))	-0.090326	0.039193	-2.304646	0.0216
ARTIK(-1)	-0.040167	0.006597	-6.088768	0.0000
R-squared	0.899249	Mean dependent var	0.024356	
Adjusted R-squared	0.896792	S.D. dependent var	1.705646	
S.E. of regression	0.547956	Akaike info criterion	1.660161	
Sum squared resid	147.7257	Schwarz criterion	1.768912	
Log likelihood	-406.1907	F-statistic	365.9451	
Durbin-Watson stat	2.119809	Prob(F-statistic)	0.000000	

Table A.12: VECM Test for first difference of EEG Data taken from O2_AV Ch.

For 512 Samples

Dependent Variable: D(O2_AV)

Method: Least Squares

Date: 11/27/06 Time: 23:16

Sample (adjusted): 8 512

Included observations: 505 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
C	-0.001800	0.028957	-0.062160	0.9505
D(O1_AV(-1))	0.126706	0.055687	2.275315	0.0233
D(O1_AV(-2))	-0.458759	0.117168	-3.915382	0.0001
D(O1_AV(-3))	0.692304	0.147868	4.681913	0.0000
D(O1_AV(-4))	-0.687259	0.144540	-4.754802	0.0000
D(O1_AV(-5))	0.389431	0.106149	3.668725	0.0003
D(O1_AV(-6))	-0.113222	0.050525	-2.240890	0.0255
D(O2_AV(-1))	1.932209	0.047911	40.32952	0.0000
D(O2_AV(-2))	-2.175236	0.094636	-22.98530	0.0000
D(O2_AV(-3))	1.853593	0.113430	16.34123	0.0000
D(O2_AV(-4))	-1.098811	0.096556	-11.38006	0.0000
D(O2_AV(-5))	0.279673	0.050469	5.541443	0.0000
ARTIK(-1)	0.008196	0.007967	1.028700	0.3041
R-squared	0.893969	Mean dependent var	-0.009307	
Adjusted R-squared	0.891382	S.D. dependent var	1.973579	
S.E. of regression	0.650436	Akaike info criterion	2.003057	
Sum squared resid	208.1489	Schwarz criterion	2.111808	
Log likelihood	-492.7720	F-statistic	345.6778	
Durbin-Watson stat	2.088841	Prob(F-statistic)	0.000000	

Table A.13: Non-Stationary Test for EEG data taken from ind. Under sus. Of ALZ.

Dependent Variable: D(ALZ1)

Method: Least Squares

Date: 09/05/06 Time: 17:31

Sample: 5000 8600

Included observations: 3601

Variable	Coefficient	Std. Error	t-Statistic	Prob.
ALZ1(-1)	-0.031843	0.003299	-9.652157	0.0000
D(ALZ1(-1))	0.777408	0.016441	47.28360	0.0000
D(ALZ1(-2))	0.156297	0.020928	7.468398	0.0000
D(ALZ1(-3))	-0.139913	0.021080	-6.637170	0.0000
D(ALZ1(-4))	-0.131843	0.021173	-6.226994	0.0000
D(ALZ1(-5))	-0.007724	0.021262	-0.363261	0.7164
D(ALZ1(-6))	0.058845	0.021164	2.780506	0.0055
D(ALZ1(-7))	0.022504	0.020642	1.090216	0.2757
D(ALZ1(-8))	-0.053368	0.020284	-2.631075	0.0085
D(ALZ1(-9))	-0.063154	0.020281	-3.113975	0.0019
D(ALZ1(-10))	-0.014821	0.020295	-0.730291	0.4653
D(ALZ1(-11))	0.025099	0.020160	1.245022	0.2132
D(ALZ1(-12))	0.016652	0.019963	0.834150	0.4043
D(ALZ1(-13))	-0.021056	0.019916	-1.057239	0.2905
D(ALZ1(-14))	-0.036441	0.015922	-2.288767	0.0222
R-squared	0.697086	Mean dependent var		-0.000434
Adjusted R-squared	0.695903	S.D. dependent var		0.217609
S.E. of regression	0.120000	Akaike info criterion		-1.398488
Sum squared resid	51.63867	Schwarz criterion		-1.372708
Log likelihood	2532.978	Durbin-Watson stat		2.001360

Table A.3: Critical Values for the Phillips-Perron Z_t Test and for the Dickey-Fuller Test Based on Estimated OLS t -Statistic

Sample Size T	Probability that $(\hat{\rho} - 1)/\hat{\sigma}_{\hat{\rho}}$ is less than entry							
	0,01	0,025	0,05	0,10	0,090	0,95	0,975	0,99

Case 1

25	-2,66	-2,26	-1,95	-1,6	0,92	1,33	1,7	2,16
50	-2,62	-2,25	-1,95	-1,61	0,91	1,31	1,66	2,08
100	-2,6	-2,24	-1,95	-1,61	0,9	1,29	1,64	2,03
250	-2,58	-2,23	-1,95	-1,62	0,89	1,29	1,63	2,01
500	-2,58	-2,23	-1,95	-1,62	0,89	1,28	1,62	2
∞	-2,58	-2,23	-1,95	-1,62	0,89	1,28	1,62	2

Case 2

25	-3,75	-3,33	-3	-2,63	-0,37	0	0,34	0,72
50	-3,58	-3,22	-2,93	-2,6	-0,4	-0,03	0,29	0,66
100	-3,51	-3,17	-2,89	-2,58	-0,42	-0,05	0,26	0,63
250	-3,46	-3,14	-2,88	-2,57	-0,42	-0,06	0,24	0,62
500	-3,44	-3,13	-2,87	-2,57	-0,43	-0,07	0,24	0,61
∞	-3,43	-3,12	-2,86	-2,57	-0,44	-0,07	0,23	0,6

Case 3

25	-4,38	-3,95	-3,6	-3,24	-1,14	-0,8	-0,5	-0,15
50	-4,15	-3,8	-3,5	-3,18	-1,19	-0,87	-0,58	-0,24
100	-4,04	-3,73	-3,45	-3,15	-1,22	-0,9	-0,62	-0,28
250	-3,99	-3,69	-3,43	-3,13	-1,23	-0,92	-0,64	-0,31
500	-3,98	-3,68	-3,42	-3,13	-1,24	-0,93	-0,65	-0,32
∞	-3,96	-3,66	-3,41	-3,12	-1,25	-0,94	-0,66	-0,33

The probability shown at the head of the column is the area in the left-hand tail.

Source: **Wayne A. Fuller, *Introduction to Statistical Time Series*, Wiley, New York, 1976, p.373**

Table A.2: Critical Values for the Phillips-Perron Z_t Test and for the Dickey-Fuller Test Based on Estimated OLS $T(\hat{\rho} - 1)$ -Statistic

Sample Size T	Probability that $T(\hat{\rho} - 1)$ is less than entry							
	0,01	0,025	0,05	0,10	0,090	0,95	0,975	0,99

Case 1

25	-11,9	-9,3	-7,3	-5,3	1,01	1,4	1,79	2,28
50	-12,9	-9,9	-7,7	-5,5	0,97	1,35	1,7	2,16
100	-13,3	-10,2	-7,9	-5,6	0,95	1,31	1,65	2,09
250	-13,6	-10,3	-8	-5,7	0,93	1,28	1,62	2,04
500	-13,7	-10,4	-8	-5,7	0,93	1,28	1,61	2,04
∞	-13,8	-10,5	-8,1	-5,7	0,93	1,28	1,6	2,03

Case 2

25	-17,2	-14,6	-12,5	-10,2	-0,76	0,01	0,65	1,4
50	-18,9	-15,7	-13,3	-10,7	-0,81	-0,07	0,53	1,22
100	-19,8	-16,3	-13,7	-11	-0,83	-0,1	0,47	1,14
250	-20,3	-16,6	-14	-11,2	-0,84	-0,12	0,43	1,09
500	-20,5	-16,8	-14	-11,2	-0,84	-0,13	0,42	1,06
∞	-20,7	-16,9	-14,1	-11,3	-0,85	-0,13	0,41	1,04

Case 3

25	-22,5	-19,9	-17,9	-15,6	-3,66	-2,51	-1,53	-0,43
50	-25,7	-22,4	-19,8	-16,8	-3,71	-2,6	-1,66	-0,65
100	-27,4	-23,6	-20,7	-17,5	-3,74	-2,62	-1,73	-0,75
250	-28,4	-24,4	-21,3	-18	-3,75	-2,64	-1,78	-0,82
500	-28,9	-24,8	-21,5	-18,1	-3,76	-2,65	-1,78	-0,84
∞	-29,5	-25,1	-21,8	-18,3	-3,77	-2,66	-1,79	-0,87

All entries in the left half of the table have standard errors less than 0,15; those in the right half, less than 0,03. The probability shown at the head of the column is the area in the left-hand tail.

Source: **Wayne A. Fuller**, *Introduction to Statistical Time Series*, Wiley, New York, 1976, p.371

Table A.14: Critical Values of Co-Integration Tests

Number Of Exp. Variable n	Critical Values for the $\hat{Z}_t$ and ADF Statistics						
	$0,1500$	$0,1250$	$0,1000$	$0,0750$	$0,0500$	$0,0250$	$0,0100$

Case 1

1	-2,2584	-2,3533	-2,4505	-2,5822	-2,7619	-3,0547	-3,3865
2	-2,7936	-2,8797	-2,9873	-3,1105	-3,2667	-3,5484	-3,8395
3	-3,2639	-3,3529	-3,4446	-3,5716	-3,7371	-3,9895	-4,3038
4	-3,6108	-3,7063	-3,8068	-3,9482	-4,1261	-4,3798	-4,672
5	-3,9438	-4,0352	-4,1416	-4,2521	-4,3999	-4,6676	-4,9897
$\Delta 1$	(-0,0232) (+0,0211)	(-0,0247) (+0,0228)	(-0,0269) (+0,0218)	(-0,0328) (+0,0347)	(-0,0439) (+0,0318)	(-0,0382) (+0,0601)	(-0,0600) (+0,0755)

Case 2

1	-2,8639	-2,9571	-3,0657	-3,1982	-3,3654	-3,642	-3,9618
2	-3,2646	-3,3513	-3,4494	-3,5846	-3,7675	-4,0217	-4,3078
3	-3,6464	-3,7306	-3,8329	-3,956	-4,1121	-4,3747	-4,7325
4	-3,9593	-4,0528	-4,1565	-4,2883	-4,4542	-4,7075	-5,0728
5	-4,2355	-4,3288	-4,4309	-4,5553	-4,7101	-4,9809	-5,2812
$\Delta 1$	(-0,0290) (+0,0186)	(-0,0261) (+0,0263)	(-0,0232) (+0,0317)	(-0,0296) (+0,0380)	(-0,0424) (+0,0304)	(-0,0389) (+0,0415)	(-0,0582) (+0,0501)

Case 3

1	-3,3283	-3,4207	-3,5184	-3,6467	-3,8	-4,0722	-4,3628
2	-3,6613	-3,74	-3,8429	-3,9754	-4,1567	-4,3854	-4,6451
3	-3,9976	-4,0808	-4,195	-4,3198	-4,4895	-4,7699	-5,0433
4	-4,2751	-4,3587	-4,4625	-4,5837	-4,7423	-5,018	-5,3576
5	-4,5455	-4,6248	-4,7311	-4,8695	-5,0282	-5,3056	-5,5849
$\Delta 1$	(-0,0259) (+0,0246)	(-0,0246) (+0,0281)	(-0,0244) (+0,0205)	(-0,0259) (+0,0301)	(-0,0350) (+0,0288)	(-0,0469) (+0,0507)	(-0,0629) (+0,0722)

Source: MacKinnon (1991), "Critical Values for Co-Integration Tests", Long-Run Economic Relationships, Oxford University Press, 267-76

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APPENDIX

A. STANDARD KALMAN FILTER

This useful tools named for the contributions of R.E.Kalman (1960,1963). The idea is to express a dynamic system in a particular form called the state-space representation. The Kalman filter is an algorithm for sequentially updating a linear projection for the system. Among other benefits, this algorithm provides a way to calculate exact finite-sample forecasts and the exact likelihood function for Gaussian ARMA processes, and to factor matrix autocovariance-generating functions or spectral densities, and to estimate vector autoregressions with coefficients that change over time. In Brain research, this algorithm is used generally to estimate the unobserved signal sources that are assumed to produce observed EEG (recordings) data.

$$Z_{t+1} = FZ_t + v_{t+1} \Leftrightarrow \begin{bmatrix} z_{1,t+1} \\ z_{2,t+1} \\ z_{3,t+1} \\ \vdots \\ \vdots \\ z_{r,t+1} \end{bmatrix} = \begin{bmatrix} F_{1,1} & F_{1,2} & \cdots & & \\ F_{2,1} & F_{2,2} & \cdots & & \\ F_{3,1} & F_{3,2} & \cdots & & \\ \vdots & \vdots & \vdots & & \\ \vdots & \vdots & \vdots & & \\ F_{r,1} & \cdots & \cdots & & \end{bmatrix} \begin{bmatrix} z_{1,t} \\ z_{2,t} \\ z_{3,t} \\ \vdots \\ \vdots \\ z_{r,t} \end{bmatrix} + \begin{bmatrix} v_{1,t+1} \\ v_{2,t+1} \\ v_{3,t+1} \\ \vdots \\ \vdots \\ v_{r,t+1} \end{bmatrix}$$

$$y_t = Ax_t + HZ_t + w_t \Leftrightarrow$$

$$\begin{bmatrix} y_{1,t} \\ y_{2,t} \\ y_{3,t} \\ \vdots \\ \vdots \\ y_{n,t} \end{bmatrix} = \begin{bmatrix} A_{1,1} & A_{1,2} & \cdots & & \\ A_{2,1} & A_{2,2} & \cdots & & \\ A_{3,1} & A_{3,2} & \cdots & & \\ \vdots & \vdots & \vdots & & \\ \vdots & \vdots & \vdots & & \\ A_{n,1} & \cdots & \cdots & & \end{bmatrix} \begin{bmatrix} x_{1,t} \\ x_{2,t} \\ x_{3,t} \\ \vdots \\ \vdots \\ x_{k,t} \end{bmatrix} + \begin{bmatrix} H_{1,1} & H_{1,2} & \cdots & & \\ H_{2,1} & H_{2,2} & \cdots & & \\ H_{3,1} & H_{3,2} & \cdots & & \\ \vdots & \vdots & \vdots & & \\ \vdots & \vdots & \vdots & & \\ H_{n,1} & H_{n,2} & \cdots & & \end{bmatrix} \begin{bmatrix} z_{1,t} \\ z_{2,t} \\ z_{3,t} \\ \vdots \\ \vdots \\ z_{r,t} \end{bmatrix} + \begin{bmatrix} w_{1,t} \\ w_{2,t} \\ w_{3,t} \\ \vdots \\ \vdots \\ w_{n,t} \end{bmatrix}$$

Let y_t denote an $(n \times 1)$ vector of variables observed at date t . A rich class of dynamic models y_t can be described in terms of a possibly unobserved $(r \times 1)$ vector Z_t known

as state vector. The state-space representation of the dynamics of y is given by the following system of equations :

$$Z_{t+1} = FZ_t + v_{t+1} \quad (\text{A1})$$

$$y_t = Ax_t + HZ_t + w_t$$

Where F , A and H are matrices of parameters of dimension $(r \times r)$, $(n \times k)$ and $(n \times r)$ respectively, and x_t is a $(k \times 1)$ vector of exogenous or predetermined variables.

Assumptions :

$$\begin{aligned} \text{(i)} \quad E(v_t v'_\tau) &= E \left\{ \begin{bmatrix} v_{1,t} \\ v_{2,t} \\ \vdots \\ v_{r,t} \end{bmatrix} \begin{bmatrix} v_{1,t} & v_{2,t} & \cdots & v_{r,t} \end{bmatrix} \right\} \quad E(v_t v'_\tau) = \begin{cases} Q & \text{for } t = \tau \\ 0 & \text{otherwise} \end{cases} \\ \text{(ii)} \quad E(w_t w'_\tau) &= E \left\{ \begin{bmatrix} w_{1,t} \\ w_{2,t} \\ \vdots \\ w_{n,t} \end{bmatrix} \begin{bmatrix} w_{1,t} & w_{2,t} & \cdots & w_{n,t} \end{bmatrix} \right\} \quad E(w_t w'_\tau) = \begin{cases} R & \text{for } t = \tau \\ 0 & \text{otherwise} \end{cases} \\ \text{(iii)} \quad E(v_t w'_\tau) &= E \left\{ \begin{bmatrix} v_{1,t} \\ v_{2,t} \\ \vdots \\ v_{r,t} \end{bmatrix} \begin{bmatrix} w_{1,t} & w_{2,t} & \cdots & w_{n,t} \end{bmatrix} \right\} \\ &\Rightarrow E(v_t w'_\tau) = \{0 \quad \text{for all } t \text{ and } \tau\} \end{aligned}$$

Assumptions about Initial Values:

$$E(v_t Z'_1) = \{0 \quad \text{for } t = 1, 2, 3, \dots, T\} \quad E(w_t Z'_1) = \{0 \quad \text{for } t = 1, 2, 3, \dots, T\}$$

$$Z_t = v_t + Fv_{t-1} + F^2 v_{t-2} + \dots + F^{t-2} v_2 + F^{t-1} Z_1 \quad \text{for } t = 2, 3, 4, \dots, T$$

$$E(v_t Z'_\tau) = 0 \quad \text{for } \tau = t-1, t-2, \dots, 1$$

$$E(w_t Z'_\tau) = 0 \quad \text{for } \tau = 1, 2, 3, \dots, T$$

$$E(w_t y'_\tau) = E \left[w_t (Ax_t + HZ_t + w_t)' \right] = 0 \quad \text{for } \tau = t-1, t-2, \dots, 1$$

$$E(v_t y'_\tau) = 0 \quad \text{for } \tau = t-1, t-2, \dots, 1$$

Derivation of Kalman Filter

$$\hat{Z}_{t+1} = \hat{E}(Z_{t+1} | Y_t)$$

$$Y_t \equiv (y'_t, y'_{t-1}, \dots, y'_1, x'_t, x'_{t-1}, \dots, x'_1)'$$

$$P_{t+1} = E \left[(Z_{t+1} - \hat{Z}_{t+1|t}) (Z_{t+1} - \hat{Z}_{t+1|t})' \right] \quad \text{associated Mean Squared Error matrix.}$$

Starting Recursion

$\hat{Z}_{1|0} = E(Z_1)$ unconditional mean. When $t=0$, there is no observed or exogenous data y or x .

$$Z_0 \sim N(\mu_0, \text{Var} Z_0)$$

$$P_{1|0} = E \left\{ [Z_1 - E(Z_1)] [Z_1 - E(Z_1)]' \right\}$$

More generally, if eigenvalues of F are all inside the unit circle, then the process for Z_t is covariance-stationary. The unconditional mean of Z_t ,

$$E(Z_{t+1}) = E(FZ_t + v_{t+1}) = FE(Z_t) + E(v_{t+1}) = FE(Z_t)$$

or

$$(I_r - F).E(Z_t) = 0 \quad \Rightarrow \quad E(Z_t) = 0 \quad \text{unique solution.}$$

The unconditional variance of Z_t ;

$$E \left[(Z_{t+1} - E(Z_{t+1})) (Z_{t+1} - E(Z_{t+1}))' \right] = E \left[(FZ_t + v_{t+1}) (Z'_t F' + v'_{t+1}) \right] = FE(Z_t Z'_t) F' + E(v_{t+1} v'_{t+1})$$

Let Σ denote the variance-covariance matrix of Z_t , thus

$$\Sigma = F \Sigma F' + Q \quad \Leftrightarrow \quad \text{vec}(\Sigma) = [I_{r \times r} - (F \otimes F)]^{-1} \cdot \text{vec}(Q)$$

Thus, in general, provided that the eigenvalues of F are inside the unit circle, the Kalman Filter iterations can be started with $\hat{Z}_{1|0} = 0$ and

$$\text{vec}(P_{1|0}) = [I_{r \times r} - (F \otimes F)]^{-1} \cdot \text{vec}(Q)$$

If some values of F are not inside the unit circle then the analyst must guess the initial value which must minimize the diagonal values of $P_{1|0}$ matrix.

Forecasting y_t :

Assumption:

Since we have assumed that x_t contains no information about Z_t beyond that contained in Y_{t-1} ,

$$\hat{E}(Z_t | x_t, Y_{t-1}) = \hat{E}(Z_t | Y_{t-1}) = \hat{Z}_{t|t-1}$$

$$\hat{y}_{t|t-1} \equiv \hat{E}(y_t | x_t, Y_{t-1}) \quad \hat{E}(y_t | x_t, Z_t) = E(Ax_t + HZ_t + w_t) = Ax_t + HZ_t$$

From the law of iterated projections:

$$\hat{y}_{t|t-1} = Ax_t + H \cdot \hat{E}(Z_t | x_t, Y_{t-1}) = Ax_t + H\hat{Z}_{t|t-1}$$

The error of this forecast is:

$$y_t - \hat{y}_{t|t-1} = Ax_t + HZ_t + w_t - Ax_t - H\hat{Z}_{t|t-1} = H(Z_t - \hat{Z}_{t|t-1}) + w_t$$

With associated MSE

$$\begin{aligned} E[(y_t - \hat{y}_{t|t-1})(y_t - \hat{y}_{t|t-1})'] &= E[(H(Z_t - \hat{Z}_{t|t-1}) + w_t)(H(Z_t - \hat{Z}_{t|t-1}) + w_t)'] \\ &= E[H(Z_t - \hat{Z}_{t|t-1})(Z_t - \hat{Z}_{t|t-1})' H'] + E(w_t w_t') \quad \text{because} \quad E[w_t(Z_t - \hat{Z}_{t|t-1})'] = 0 \\ E[(y_t - \hat{y}_{t|t-1})(y_t - \hat{y}_{t|t-1})'] &= HP_{t|t-1}H' + R \end{aligned}$$

Updating the Inference About Z_t : Correction

The inference about the current value of Z_t is updated on the basis of the observation of y_t to produce

$$\hat{Z}_{t|t} = \hat{E}(Z_t | y_t, x_t, Y_{t-1}) = \hat{E}(Z_t | Y_t)$$

$$\hat{Z}_{t|t} = \hat{Z}_{t|t-1} + \left\{ E[(Z_t - \hat{Z}_{t|t-1})(y_t - \hat{y}_{t|t-1})'] \right\} \times \left\{ E[(y_t - \hat{y}_{t|t-1})(y_t - \hat{y}_{t|t-1})'] \right\}^{-1} \times (y_t - \hat{y}_{t|t-1})$$

But

$$\begin{aligned} E[(Z_t - \hat{Z}_{t|t-1})(y_t - \hat{y}_{t|t-1})'] &= E\left\{ [Z_t - \hat{Z}_{t|t-1}] \mathbb{I} [H(Z_t - \hat{Z}_{t|t-1}) + w_t] \right\} \\ &= E\left\{ [Z_t - \hat{Z}_{t|t-1}] \mathbb{I} [Z_t - \hat{Z}_{t|t-1}] H + [Z_t - \hat{Z}_{t|t-1}] w_t' \right\} \\ &= P_{t|t-1} H' \end{aligned}$$

Thus

$$\hat{Z}_{t|t} = \hat{Z}_{t|t-1} + P_{t|t-1} H' (HP_{t|t-1} H' + R)^{-1} (y_t - Ax_t - H\hat{Z}_{t|t-1})$$

The MSE associated with this updated projection $P_{t|t}$:

$$\begin{aligned} P_{t|t} &\equiv E \left[(Z_t - \hat{Z}_{t|t}) (Z_t - \hat{Z}_{t|t})' \right] \\ &= E \left[(Z_t - \hat{Z}_{t|t-1}) (Z_t - \hat{Z}_{t|t-1})' \right] - \left\{ E \left[(Z_t - \hat{Z}_{t|t-1}) (y_t - \hat{y}_{t|t-1})' \right] \right\} \times \left\{ E \left[(y_t - \hat{y}_{t|t-1}) (y_t - \hat{y}_{t|t-1})' \right] \right\}^{-1} \\ &\quad \times \left\{ E \left[(y_t - \hat{y}_{t|t-1}) (Z_t - \hat{Z}_{t|t-1})' \right] \right\} \\ &= P_{t|t-1} - P_{t|t-1} H' (HP_{t|t-1} H' + R)^{-1} HP_{t|t-1} \end{aligned}$$

Producing a Forecast of Z_{t+1} :

$$\begin{aligned} \hat{Z}_{t+1|t} &= \hat{E}(Z_{t+1}|Y_t) = F \cdot \hat{E}(Z_t|Y_t) + \hat{E}(v_{t+1}|Y_t) \\ &= F\hat{Z}_{t|t} + 0 \end{aligned} \tag{A.2}$$

Replacing $\hat{Z}_{t|t}$ gives:

$$\hat{Z}_{t+1|t} = F\hat{Z}_{t|t-1} + FP_{t|t-1} H' (HP_{t|t-1} H' + R)^{-1} (y_t - Ax_t - H\hat{Z}_{t|t-1})$$

Kalman Gain Matrix is the coefficient Matrix and is denoted by K_t :

$$K_t = FP_{t|t-1} H' (HP_{t|t-1} H' + R)^{-1}$$

By the way we can rewrite $\hat{Z}_{t+1|t}$:

$$\hat{Z}_{t+1|t} = F\hat{Z}_{t|t-1} + K_t (y_t - Ax_t - H\hat{Z}_{t|t-1})$$

The MSE of this forecast can be found from (A.2) and the state equation (A.1):

$$\begin{aligned}
P_{t+1|t} &= E \left[(Z_{t+1} - \widehat{Z}_{t+1|t}) (Z_{t+1} - \widehat{Z}_{t+1|t})' \right] \\
&= E \left[(FZ_t + v_{t+1} - F\widehat{Z}_{t|t}) (FZ_t + v_{t+1} - F\widehat{Z}_{t|t})' \right] \\
&= E \left[(FZ_t - F\widehat{Z}_{t|t} + v_{t+1}) (Z_t' F' - \widehat{Z}_{t|t}' F' + v_{t+1}') \right] \\
&= FE \left[(Z_t - \widehat{Z}_{t|t}) (Z_t - \widehat{Z}_{t|t})' \right] F' + E[v_{t+1} v_{t+1}'] \\
&= FP_{t|t} F' + Q
\end{aligned}$$

Replacing $P_{t|t}$ gives us:

$$\begin{aligned}
P_{t+1|t} &= F \left[P_{t|t-1} - P_{t|t-1} H' (HP_{t|t-1} H' + R)^{-1} HP_{t|t-1} \right] F' + Q \\
\widehat{y}_{t+1|t} &\equiv \widehat{E}(y_{t+1} | x_{t+1}, Y_t) = Ax_{t+1} + H\widehat{Z}_{t+1|t}
\end{aligned}$$

With associated MSE,

$$E \left[(y_{t+1} - \widehat{y}_{t+1|t}) (y_{t+1} - \widehat{y}_{t+1|t})' \right] = HP_{t+1|t} H' + R$$

B ARCH/GARCH

If a random variable y_t is drawn from the conditional density $f(y_t | y_{t-1})$, the forecast of today's value based upon the past information, under standard assumptions, is simply $E(y_t | y_{t-1})$, which depends upon the value of the conditioning variable y_{t-1} . The variance of this one-period forecast is given by $V(y_t | y_{t-1})$. Such an expression recognizes that the conditional forecast variance depends upon past information and therefore be a random variable. For conventional econometric models, however, the conditional variance does not depend upon y_{t-1} . But the excellent study of Engle (1982) proposes a class of models where the variance does depend upon the past and has proved their usefulness in economics and latter it is also widely accepted in research areas concerning "dynamical inverse problems". In this part of our study we will only mention theoretical foundations of this idea, the process' properties and its generalized version.

Consider initially the first-order autoregression

Where ε is white noise with $V(\varepsilon) = \sigma^2$. The conditional mean of y_t is $\mathcal{Y}_{t-1}$ while the unconditional mean is zero. Clearly, the vast improvement in forecasts due to time-series models stems from the use of the conditional mean. The conditional variance of y_t is σ^2 while the unconditional variance is $\frac{\sigma^2}{1 - \gamma^2}$. For real processes one might expect better forecast intervals if additional information from the past were allowed to effect the forecast variance; a more general class of models seems desirable.

The standart approach of heteroscedasticity is to introduce an exogenous variable x_t which predicts the variance. With a known zero mean, the model might be $y_t = \varepsilon_t x_{t-1}$

Where again $V(\varepsilon) = \sigma^2$. The variance of y_t is simply $V(y_t) = \sigma^2 x_{t-1}^2$, and, therefore, the forecast interval depends upon the evolution of an exogenous variable. This standard solution to the problem seems unsatisfactory, as it requires a specifications of the causes of the changing variance, rather than recognizing that both conditional means and variances may jointly evolve over time. Perhaps because of this difficulty, heteroscedasticity corrections are rarely condisered in time-series data.

A model which allows the conditional variance to depend on the past realization of the series is the bilinear model described by Granger and Andersen. A simple case is $y_t = \varepsilon_t y_{t-1}$

The conditional variance is now $\sigma^2 y_{t-1}^2$. However, the unconditional variance is either zero or infinity, which makes this an unattractive formulation, although slight generalizations avoid this problem.

A preferable model is

$$y_t = \varepsilon_t h_t^{1/2}$$

$$h_t = \alpha_0 + \alpha_1 y_{t-1}^2$$

With $V(\varepsilon_t)=1$ This is an example of what will be called an autoregressive conditional heteroskedasticity (ARCH) model, adding the assumption of normality, it can be more directly expressed in terms of ψ_t , the information set available at time t

$$y_t|\psi_{t-1} \sim N(0, h_t) \quad (\text{B.1})$$

$$h_t = \alpha_0 + \alpha_1 y_{t-1}^2 \quad (\text{B.2})$$

The variance function can be expressed more generally as

$$h_t = h(y_{t-1}, y_{t-2}, y_{t-3}, \dots, y_{t-p}, \alpha) \quad (\text{B.3})$$

where p is the order of the ARCH process and α is a vector of unknown parameters. The ARCH regression model is obtained by assuming that the mean of y_t is given as $x_t\beta$, a linear combination of lagged endogenous and exogenous variables included in the information set ψ_{t-1} with β a vector of unknown parameters. Formally,

$$y_t|\psi_{t-1} \sim N(0, h_t)$$

$$h_t = h(\varepsilon_{t-1}, \varepsilon_{t-2}, \dots, \varepsilon_{t-p}, \alpha) \quad (\text{B.4})$$

$$\varepsilon_t = y_t - x_t\beta$$

The variance function can be further generalized to include current and lagged x 's as these also enter the information set. The h function then becomes

$$h_t = h(\varepsilon_{t-1}, \varepsilon_{t-2}, \dots, \varepsilon_{t-p}, x_t, x_{t-1}, \dots, x_{t-p}, \alpha) \quad (\text{B.5})$$

Or simply

$$h_t = h(\psi_{t-1}, \alpha)$$

In particular, if the h function factors into

$$h_t = h_\varepsilon(\varepsilon_{t-1}, \dots, \varepsilon_{t-p}, \alpha) h_x(x_t, \dots, x_{t-p})$$

the two types of heteroscedasticity can be dealt with sequentially by first correcting for the x component and then fitting the ARCH model on the transformed data.

The ARCH regression model in (B.4) has a variety of characteristics which make it attractive for econometric applications. Econometric forecasters have found that their ability to predict the future varies from period to another. Mcnees suggests that, “the inherent uncertainty or randomness associated with different forecast periods seems to vary widely over time.” He also documents that, “large and small errors tend to cluster together (in contiguous time periods).” This analysis immediately suggests the usefulness of the ARCH model where the underlying forecast variance may change over time and is predicted by past forecast errors. The results presented by McNees also show some serial correlation during the episodes of large variance.

A second example is found in monetary theory and theory of finance. By the simplest assumptions, portfolios of financial assets are held as functions of the expected means and variances of the rates of return. Any shifts in asset demand must be associated with changes in expected means and variances of the rates of return. If the mean is assumed to follow a standard regression or time-series model, the variance is immediately constrained to be constant over time. The use of an exogenous variable to explain changes in variance is usually not appropriate.

A third interpretation is that the ARCH regression model is an approximation to a more complex regression which has non-ARCH disturbances. The ARCH specification might then be picking up the effect of variables omitted from the estimated model. The existence of an ARCH effect would be interpreted as evidence of misspecification, either by omitted variables or through structural change. If this is the case, ARCH may be a better approximation to reality than making standard assumptions about the disturbances, but trying to find the omitted variable or determine the nature of the structural change would be even better.

B.1 The Likelihood Function

Suppose y_t is generated by an ARCH process described in equations (B.1) and (B.3). The properties of this process can easily be determined by repeated application of the relation $Ex = E(Ex|\psi)$. The mean of y_t is zero and all auto-covariances are zero. The unconditional variance is given by $\sigma^2_t = Ey^2_t = Eh_t$. For many functions h and values of α , the variance is independent of t . Under such conditions, y_t is covariance stationary; a set of sufficient conditions for this is derived below.

Although the process defined by (B.1) and (B.3) has all observations conditionally distributed, the vector of y is not jointly normally distributed. The joint density is the product of all the conditional densities and, therefore, the log likelihood is the sum of the conditional normal log likelihoods corresponding to (B.1) and (B.3). Let l be the average log likelihood and l_t be the log likelihood of the t th observation and T the sample size. Then

$$l = \frac{1}{T} \sum_{t=1}^T l_t \quad (\text{B.6})$$

$$l_t = -\frac{1}{2} \log h_t - \frac{1}{2} y^2_t / h_t$$

Apart from some constants in the likelihood.

To estimate the unknown parameters α , the likelihood function can be maximized. The first-order conditions are

$$\frac{\partial l_t}{\partial \alpha} = \frac{1}{2h_t} \frac{\partial h_t}{\partial \alpha} \left(\frac{y^2_t}{h_t} - 1 \right) \quad (\text{B.7})$$

And the Hessian is

$$\frac{\partial^2 l_t}{\partial \alpha \partial \alpha'} = -\frac{1}{2h_t^2} \frac{\partial h_t}{\partial \alpha} \frac{\partial h_t}{\partial \alpha'} \left(\frac{y_t^2}{h_t} \right) + \left[\frac{y_t^2}{h_t} - 1 \right] \frac{\partial}{\partial \alpha'} \left[\frac{1}{2h_t} \frac{\partial h_t}{\partial \alpha} \right] \quad (\text{B.8})$$

The conditional expectation of the second term, given ψ_{t-m-1} , is zero, and of the last factor in the first, is just one. Hence, the information matrix, which is simply the negative expectation of the Hessian averaged over all observations, becomes

$$\xi_{\alpha\alpha} = \sum_t \frac{1}{2T} E \left[\frac{1}{h_t^2} \frac{\partial h_t}{\partial \alpha} \frac{\partial h_t}{\partial \alpha'} \right] \quad (\text{B.9})$$

Which is consistently estimated by

$$\hat{\xi}_{\alpha\alpha} = \frac{1}{T} \sum_t \left[\frac{1}{2h_t^2} \frac{\partial h_t}{\partial \alpha} \frac{\partial h_t}{\partial \alpha'} \right] \quad (\text{B.10})$$

If the h function is p th order linear (in the squares), so that it can be written as

$$h_t = \alpha_0 + \alpha_1 y_{t-1}^2 + \dots + \alpha_p y_{t-p}^2 \quad (\text{B.11})$$

Then the information matrix and gradient have a particularly simple form. Let

$$z_t = (1, y_{t-1}^2, \dots, y_{t-p}^2) \text{ and } \alpha' = (\alpha_0, \alpha_1, \dots, \alpha_p)$$

So that (B.11) can be rewritten as

$$h_t = z_t \alpha \quad (\text{B.12})$$

The gradient then becomes simply

$$\frac{\partial l}{\partial \alpha} = \frac{1}{2h_t} z_t \left(\frac{y_t^2}{h_t} - 1 \right) \quad (\text{B.13})$$

And the estimate of the information matrix

$$\hat{\xi}_{\alpha\alpha} = \frac{1}{2T} \sum_t \left[\frac{z_t' z_t}{h_t^2} \right] \quad (\text{B.14})$$

B.2 Distribution of the First-Order Linear ARCH process

The simplest and often very useful ARCH model is the first-order linear model given by (B.1) and (B.2). A large observation for y_t will lead to a large variance for the next period's distribution, but the memory is confined to one period. If $\alpha_1 = 0$, of course y_t will be Gaussian white noise and if it is a positive number, successive observations will be dependent through higher-order moments. As shown below, if α_1 is too large, the variance of the process will be infinite.

To determine the conditions for the process to be stationary and to find the marginal distribution of the y_t 's, a recursive argument is required. The odd moments are immediately seen to be zero by symmetry and the even moments are computed using the following theorem. In all cases it is assumed that the process begins indefinitely far in the past with $2r$ finite initial moments.

Theorem 1: For integer r , the $(2r)^{th}$ moment of a first-order linear ARCH process with $\alpha_0 > 0, \alpha_1 \geq 0$ exists if, and only if,

$$\alpha_1^r \prod_{j=1}^r (2j-1) < 1$$

The theorem is easily used to find the second and fourth moments of a first-order process. Letting $w_t = (y_t^4, y_t^2)'$

$$E(w_t | \mathcal{F}_{t-1}) = \begin{pmatrix} 3\alpha_0^2 \\ \alpha_0 \end{pmatrix} + \begin{pmatrix} 3\alpha_1^2 & 6\alpha_0\alpha_1 \\ 0 & \alpha_1 \end{pmatrix} w_{t-1}$$

The condition for the variance to be finite is simply that $\alpha_1 < 1$, while to have a finite fourth moment it is also required that $3\alpha_1^2 < 1$. If the conditions are met, the moments can be computed as

$$E(w_t) = \left[\begin{array}{c} \left[\frac{3\alpha_0^2}{(1-\alpha_1)^2} \right] \left[\frac{1-\alpha_1^2}{1-3\alpha_1^2} \right] \\ \frac{\alpha_0}{1-\alpha_1} \end{array} \right] \quad (\text{B.15})$$

The lower element is the unconditional variance, while the upper product gives the fourth moment. The first expression in square brackets is three times the squared variance. For $\alpha_1 \neq 0$, the second term is strictly greater than one implying a fourth moment greater than that of a normal random variable.

The first-order ARCH process generates data with fatter tails than the normal density. Many statistical procedures have been designed to be robust to large errors, but to the as we know, none of this literature has made use of the fact that temporal clustering of outliers can be used to predict their occurrence and minimize their effects. This is exactly the approach taken by the ARCH model.

B.3 General ARCH process

The conditions for a first-order linear ARCH process to have a finite variance and, therefore, to be covariance stationary can directly be generalized for p th-order process.

Following from Engle (1982) we can write this theorem:

Theorem 2: The p th order linear ARCH process, with $\alpha_0 > 0, \alpha_1, \dots, \alpha_p \geq 0$..., is covariance stationary if, and only if, the associated characteristic equation has all roots outside the unit circle. The stationary variance is given by $E(y_t^2) = \frac{\alpha_0}{\left(1 - \sum_{j=1}^p \alpha_j\right)}$

Although the p th order linear model is a convenient specification, it is likely, that other formulations of the variance model may be more appropriate for particular applications. Two simple alternatives are the exponential and absolute value forms:

$$h_t = e^{(\alpha_0 + \alpha_1 y_{t-1}^2)} \quad (\text{B.16})$$

$$h_t = \alpha_0 + \alpha_1 |y_{t-1}| \quad (\text{B.17})$$

These provide an interesting contrast. The exponential form has the advantage that the variance is positive for all values of α , but it is not difficult to show that data generated from such a model have infinite variance for any value of $\alpha_1 \neq 0$. The implications of this deserve further study. The absolute value form requires both parameters to be positive, but can be shown to have finite variance for any parameter values.

In order to find estimation results which are more general than the linear model, general conditions on the variance model will be formulated and shown to be implied for the linear process.

Let ζ_t be $p \times 1$ random vector drawn from the sample space Θ , which has elements $\zeta_t' = (\zeta_{t-1}, \zeta_{t-2}, \dots, \zeta_{t-p})$. For any ζ_t let ζ_t^* be identical, except that m th element has been multiplied by -1, where m lies between 1 and p .

Definition: The ARCH process defined by (B.1) and (B.3) is symmetric if

- (a) $h(\zeta_t) = h(\zeta_t^*)$ for any m and $\zeta_t \in \Theta$
- (b) $\frac{\partial h(\zeta_t)}{\partial \alpha_i} = \frac{\partial h(\zeta_t^*)}{\partial \alpha_i}$ for any m and $\zeta_t \in \Theta$
- (c) $\frac{\partial h(\zeta_t)}{\partial \zeta_{t-m}} = \frac{-\partial h(\zeta_t^*)}{\partial \zeta_{t-m}}$ for any m and $\zeta_t \in \Theta$

All the functions described have been symmetric. This condition is the main distinction between mean and variance models.

Another characterization of general ARCH models is in terms of regularity conditions.

Definition: The ARCH model defined by (B.1) and (B.3) is regular if

- (a) $\min h(\zeta_t) \geq \delta$ for some $\delta > 0$ and $\zeta_t \in \Theta$

$$(b) \quad E \left(\left\| \frac{\partial h(\zeta_t)}{\partial \alpha_i} \right\| \left\| \frac{\partial h(\zeta_t)}{\partial \zeta_{t-m}} \right\| \middle| \psi_{t-m-1} \right) \text{ exists for all } i, m, t$$

The first portion of the definition is very important and easy to check, as it requires the variance always to be positive. This eliminates, for example log-log autoregression. The second portion is difficult to check in some cases, yet should generally be true if the process is stationary with bounded derivatives, since conditional expectations are finite if unconditional ones are.

Theorem 3: The p th order linear ARCH model satisfies the regularity conditions, if $\alpha_0 > 0, \alpha_1, \dots, \alpha_p \geq 0$

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