Aspects of Sexual Function in people with Epilepsy

Susan Duncan

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ABSTRACT OF THESIS

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ABSTRACT OF THESIS: Using a validated questionnaire {Frenken and Vennix Sexuality Experience Scales (SES) 1981} the aims of this study are to -:

1. Examine the attitudes to sexual morality, and premarital sexual experience of people with epilepsy compared with a randomly recruited control group.
2. To ascertain whether people with epilepsy differ in their openness to psychosexual stimulation, in particular their arousability by erotic imagery, and their feelings upon being exposed to such imagery.
3. To compare those with epilepsy's enjoyment of sexual interaction and preferred frequency of such activity compared with the control group, employing .
4. To examine the effects of antiepileptic drug (AED) levels on arousability
5. To determine the effects, if any, of seizure type, frequency, and age at onset of epilepsy on indices of arousability and preferred frequency of intercourse.
6. To examine the relationship, if any, between free testosterone and preferred frequency of intercourse
7. To examine the effects in men of individual AEDs on sex hormone binding globulin (SHBG), total (TT) and free testosterone (FT) levels, and in women to study the effects of AEDs on SHBG,TT,FT.

159 women and 153 men with epilepsy receiving AEDs (treated group) along with 36 women and 34 men not receiving any AED therapy (untreated group) were recruited from a specialist epilepsy clinic. In addition 48 healthy women and 45 healthy men were recruited to act as controls. All were administered the SES 1, which examines attitudes to sexual morality, and SES 2 openness to psychosexual stimulation. Only those in stable heterosexual relationships were asked to complete SES 3 the attraction to sexual partner scale, and SES 4 attraction to marriage scale. Each SES scale is subdivided into subscales designed to examine in greater detail some aspect of the scale. Blood was withdrawn at the same time from 183 of the men and 131 of the women for AED. androgen, and SHBG levels.

Men in the treated group had a significantly higher mean SES 1 (unweighted) score and MS (sexual morality within marriage) subscale score than the controls suggesting they espoused a stricter sexual morality. Multiple regression showed further education had a significant predictive effect. Both untreated and treated groups had significantly higher mean SES 2 (unweighted) and AE (arousal by erotic imagery) subscale scores. In addition the untreated group had a higher mean IS (interpersonal sexual attraction) subcale score than the control group. This implies that overall the men with epilepsy were less open to psychosexual stimulation, and less aroused by erotic love scenes. Multiple regression showed further education to have a predictive effect for AE subscale scores. There were no significant differences between the groups on SES 3 or its subscales or on SES 4 (unweighted). Men in the treated group had a significantly lower mean MS (marital satisfaction) subcale score than controls suggesting they found their marriages more satisfactory. Those men with primary generalised epilepsy (PGE) and complex partial seizures (PGE) had significantly higher mean MS (sexual morality within marriage) scores than controls, and the PGE and undefined seizure type group had significantly higher AE (arousal by erotic imagery) scores than controls. Linear regression failed to demonstrate a significant relationship between age at onset of epilepsy, and seizure frequency and AE (arousal by erotic imagery) subscale or FI (preferred frequency of intercourse) subscale scores. Neither was there any relationship between AED levels and AE (arousal by erotic imagery) subscale scores. Men taking enzyme inducing AEDs and combinations of AEDs had significantly raised SHBG and TT levels than controls and untreated men. There were no significant differences in calculated FT between controls, untreated and men taking AEDs.

The female treated group had significantly higher SES 1 (unweighted) SS (sexual socialisation), and MS subcale scores compared with controls, with multiple regression showing further education and religious affiliation to have predictive effects. The female treated group also had higher mean SES 2 (unweighted) and AE subcale scores than controls implying treated were less open to psychosexual stimulation, they also expressed a greater sexual attraction to their partner SES 3 and greater satisfaction with their marriage SES 4. Women with PGE and CPS espoused a stricter morality within marriage, but seizure type had no effect on SES 3 or 4 and their subscales. There was no significant relationship between AED levels seizure, frequency and age at onset of epilepsy and AE or FI subscale scores. Linear regression showed a negative relationship between FI scores and FT levels of the treated group. Women receiving enzyme inducing AEDs had raised SHBG and TT, but AED therapy did not significantly effect FT levels in these women.

Both control groups had a high proportion of individuals who received further education, and there were significant age differences between the control and treated groups. Both of these parameters are known to affect SES scores. In addition standard deviations were wide in some of the SES scores. Overall it would appear that people with epilepsy living in the community, although more "moral" in outlook are no less likely to achieve satisfactory sexual relationships.

The study presented below was undertaken during my tenure as a clinical research fellow in the Department of Medicine and Therapeutics at the Western Infirmary Glasgow. I declare that both Ms Jackie Blacklaw and I collected the data. I was solely responsible for its transcription onto a standard personal computer, for the subsequent statistical analysis, and for the writing of this thesis.

Susan Duncan

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For thee the fates severely kind ordain
A cool suspension from passion and from pain
Thy life a long dead calm of quiet repose
No pulse that riots no blood that glows.

Peter Abelard (1079 - 1142)

" The degree and nature of a man's sexuality pervades the loftiest reaches of his intellect "

Friedrich Nietzsche (1844 - 1900)

" Sex is a matter of opportunity"

Gore Vidal (1925 -)

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LIST OF ABBREVIATIONS USED

AED	Antiepileptic Drug
ANOVA	One Way Analysis of Variance
STD	Standard Deviation
SES	Sexuality Experience Scale
VPA	Sodium Valproate
CBZ	Carbamazepine
PHT	Phenytoin
FT	Free Testosterone
TT	Total Testosterone
DHEAS	Dehydroepiandosterone Sulphate
AND	Androstenedione
FSH	Follicle Stimulating Hormone
LH	Luteinising Hormone
SHBG	Sex Hormone Binding Globulin
NPT	Nocturnal Penile Tumescence
ILEA	International League Against Epilepsy
CPS	Complex Partial Seizures
PGE	Primary Generalised Epilepsy
CONT	Control
MS	Sexual Morality within Marriage
PS	Premarital Sexual Experience
PA	Premarital Sexual Attitude
IS	Interpersonal Sexual Attraction
EE	Evaluation of Exposure to Erotic Imagery
AE	Arousal by Erotic Imagery
EP	Enjoyment Potential of Sexual Interaction
OE	Orgasm Adequacy During Intercourse and Its Evaluation
II	Inhibition During Intercourse
FI	(Preferred Frequency) of Sexual Intercourse
LF	Length of Foreplay
MS	Marital Satisfaction
AE	Attitude Towards Extramarital Involvement
ES	Evaluation of Partner as Sexual Partner
SS	Sexual Socialisation Morality
OCP	Oral Contraceptive Pill
FAI	Free Androgen Index
NES	None Epileptic Seizure

INTRODUCTION AND LITERATURE REVIEW

HISTORICAL VIEWS OF SEXUALITY AND EPILEPSY

Sexuality and epilepsy have been intimately linked in the minds of physicians and laymen since ancient times. The oldest written system of medicine in the world, the Ayurveda of India opined that epilepsy could be caused by sexual excess (Manyam 1992). Hippocrates wrote that both complete abstinence from and, excessive indulgence in intercourse, could cause epilepsy and described the orgasm as a form of seizure (Tempkin 1971 p32). Galen stated that seizures often occurred in girls who had intercourse at too early an age (Tempkin 1971 p32), a particularly interesting observation in the light of modern evidence linking sexual abuse with non epileptic seizures (Liske et al 1964 ; Standage 1975 ; Goodwin et al 1979 ; Gross 1979 ; Desai et al 1982).

In 1892 Von Krafft-Ebbing wrote

"Moreover in many epileptics the sexual instinct is very intense. For the most part, it is satisfied by masturbation, now and then by attacks on children and by pederasty. Perversion of the instinct with perverse sexual acts seems to be infrequent".

The belief that people with epilepsy were possessed of unbridled sexual desire was the rationale behind the introduction of bromides in the second half on the nineteenth century. These compounds had been noted to cause impotence and it was reasoned that a reduction in the libido would lead to a diminution in the number of seizures (Seiveking 1857; Gowers 1885). Other members of the profession, convinced that

masturbation was the root of all epilepsy advocated castration and cliterectomy (Tempkin 1971, page 231).

Epilepsy has always carried a stigma. In ancient times freemen with the condition were not permitted to become priests, and slaves found to have seizures were returned to their former masters and a refund demanded. The ancient belief that epilepsy was a sign of demonic possession led the church to forbid people with epilepsy from receiving holy communion in medieval France (Temkin 1971 p 143), and continued well into the sixteenth century. With the ascent of the nation state some countries notably Sweden (Alstrom 1950) and the USA (Lennox et al 1953) passed laws forbidding people with the condition to marry unless they could demonstrate that their seizures were the result of some external agency, and thus unlikely to be transmitted to their offspring. For the same reason it was illegal for people with idiopathic epilepsy to immigrate to the USA until 1965 (Lehman 1993).

People who bear a stigma have a spoiled identity. They are seen as other by the rest of society, which tends to confer upon such individuals undesirable characteristics. These are then used to deny the victim the same rights and opportunities as their contemporaries enjoy (Goffman 1969). Krafft-Ebbing could be interpreted in such a light. Writing in an age that regarded mental illness as a form of moral inadequacy, and masturbation as the highway to innumerable ills ranging from seizures to ugliness he reported cases that confirmed the beliefs of the society in which he lived.

No century exists in isolation from the preceding one, and there is little doubt that some of the misconceptions about the nature of epilepsy and its effects on the sufferers behaviour have continued into our own. As recently as 1949 13% of the American public considered epilepsy a form of insanity, and 24% said they would not like their children to play with children who suffered seizures. By 1979 these figures had improved to 3% and 6% respectively, however in the same year 18% of those surveyed said they would object to one of their children marrying a person with epilepsy (Caveness et. al. 1980). Two surveys carried out in Australia found physicians believed people with epilepsy were more likely to lose time off work and be less reliable than their peers (Beran et. al. 1981; Beran et. al. 1983)

The belief that epilepsy is associated with abnormal sexuality has persisted with publications linking the condition with homosexuality (Kolarsky et. al. 1967; Taylor 1969), fetishism (Mitchell et. al. 1954; Hunter et. al. 1963; Hooshmand et. al. 1969; Epstein 1961), transvestism (Petritzer et. al. 1955; Davies et. al. 1960; Hunter et. al. 1963), transexualism (Hoenig et. al. 1979), sado-masochism (Kolinarsky et. al. 1967; Taylor 1969), and exhibitionism (Hooshmand et. al. 1969). It is interesting to note that since 1967 there have been no reports of or studies seeking to link complex partial seizures and homosexuality. A fascinating example of culture influencing medicine.

SEXUAL SEIZURES

The reason why epilepsy has been associated with abnormal or unbridled sexuality may lie in post-ictal automatisms which can take the form of masturbatory movements and pelvic thrusting. Von Krafft-Ebbing reported the cases of three men who masturbated or exposed themselves during attacks and had no memory for their acts. He also reported a woman who during a seizure would raise her garments and make "lascivious movements" with her body.

Freemon and Nevis (1969) described the case of a man whose seizures began with an itch in the perineum then progressed to him manipulating his genitals, he was found to have a hypoplastic left middle cerebral artery. Hooshmand and Brawley (1969) reported the cases of two men who exposed themselves during seizures with no recollection of the event. One had spike activity on EEG recording over the frontal area, the other was found to have an astocytoma involving both temporal and frontal lobes on the same side. Currier (Currier et. al. 1971) reported three cases, all women, who suffered from complex partial seizures (CPS) each of whom exhibited sexual automatisms. Two of these women had abnormal surface EEGs with spike and wave abnormalities over the left temporal lobe. The third had a dysontogenetic cyst in her hypothalamus. Gautier-Smith (1980) described three cases all of whom were female who reported sexual feelings, or a desire for intercourse as part of their complex partial seizures. One had no demonstrable EEG abnormality, the other 2 showed temporal lobe foci.

In 1983 Spencer and colleagues (Spencer et. al. 1983) studied sixty-one patients with CPS fourteen of which had frontal lobe foci demonstrated either by depth electrode studies or by the presence of a structural abnormality. Four out of these fourteen patients had sexual automatisms. The authors stated that no patient had sexual automatisms if the seizure focus originated other than in the frontal lobe. Three of these patients were male one female. Thus there are remarkable few cases in the literature and it appears that seizures which give rise to such automatisms may well originate in the frontal rather than the temporal lobes. The potential for social embarrassment caused by these automatisms is immediately apparent, and it is obvious why they made such an impression on the Victorians. That potential still exists and reluctance on the part of family and friends to report such incidents may account for their comparative rarity in modern literature.

Seizures in which feelings of sexual arousal or orgasm are experienced usually as part of the aura have also been reported. Remillard and colleagues (Remillard et. al. 1983) reported 12 women all of whom reported feelings of sexual excitement often associated with vulvovaginal discharge, orgasm or the desire to have sexual intercourse. The mean age of these women was 37 years with a mean age of onset of 27 years. Two were found to have temporal lobe gliosis, one a cerebral infarct, one an astrocytoma, another postencephalitic epilepsy, three were of unknown aetiology, another had post traumatic seizures. In seven the EEG was right or predominantly right sided in three left or predominantly left-sided, and in the remaining two the EEG did not reveal discharges. Remillard reviewed the literature on the subject including the paper by Gautier-Smith

and Currier pointing out that there were only 11 such cases in the literature (1970) in which sexual arousal or orgasm was reported as part of a seizure. All of these cases were women, leading Remillard to suggest that the neural organisation of the temporal lobes of men and women differed. Another possible explanation is that such cases are more likely to be reported by a predominantly male profession who grew up in a society which disapproved of women being openly sexual.

HYPOSEXUALITY AND EPILEPSY

Despite these accounts of sexual feelings associated with complex partial seizures the last fifty years has seen a number of papers citing hyposexuality as an accompaniment of complex partial seizures (CPS). Gastuat and Collomb's paper " Etude du comportement sexuel chez les epileptiques psychomoteur " published in 1954 is usually cited in the literature as being the first report of the phenomenon. A close reading of Krafft-Ebbing, however, shows that he had observed in some epileptics an "indifference" to sex between seizures. This observation is at the end of the paragraph quoted above, given that it is preceded by pederasty and masturbation it is unsurprising that it is frequently overlooked.

Gastaut and Collomb studied a group of 36 men and women suffering from complex partial seizures, most of whom were resident in institutions. They received either phenobarbitone or phenytoin as therapy. Of these only 9 were considered to have a normal sexual interest. The remainder exhibited a global diminution in sexual activities, with infrequent or absent sexual contacts, dreams, and autoerotic behaviour. Moreover Gastaut and Collomb observed that those who were hyposexual had nearly all developed CPS before puberty. There was no relationship between seizure frequency and sexual function.

In 1967 Kolarsky et. al. studied 89 men with CPS. The aim of the paper was to establish a link between temporal lobe damage and sexual "deviation ". Each man underwent a

detailed interview and physical examination and the results were rated blind by a panel of neurologists and experts in sexual deviation. 16 of these men were considered to be hyposexual, but the authors then point out only 4 of them masturbated less than once a month, calling into question the extent of their sexual indifference. In addition to this Kolarsky and his colleagues stated the presence of a "deviation " could be "inferred" from the patient's manner, and further that their suspicion was aroused if an individual did not give an adequate explanation for not indulging in what they defined as normal sexual behaviour.

Several studies have been performed in patients being considered for surgical treatment of their seizures. Taylor (1969) reported a study of 100 patients, 63 men and 37 women who were being evaluated prior to surgery. He found 49% of the men and 60% of the women were married, and noted a trend for those with early onset disease to be less likely to marry. They assessed sexual interest on a five point scale with 1 indicating at least one sexual outlet per month and a positive attitude to sex, courtship, and marriage, through to 5 which meant the individual had abandoned normal sexual outlets, was impotent, or frigid, or was considered " deviant". They found over 50 patients rated scores of 3,4 or 5. Those with early onset of CPS were significantly more likely to exhibit poor sexual adjustment. Taylor points out that most of the group, particularly those from institutional backgrounds had received inadequate sexual education, although

only 2 were completely ignorant, and it is clear that some of the patients had not developed the social skills required to make more than superficial relationships. Post operation 22 of the patients who had scores of 3,4 or 5 improved, whilst some 14 worsened. Seizure relief was associated with improvement in sexual adjustment, and social adjustment. The finding of mesial temporal sclerosis at operation as opposed to late-acquired lesions was also associated with improvement in sexual adjustment. AED therapy is not described anywhere in this paper.

Blumer et. al. (1967) found that of 21 patients who underwent temporal lobectomy 11 were hyposexual. They interviewed their patients and sought information about frequency of sexual intercourse, autosexual behaviour, erotic dreams and fantasies. They discovered that impotence always co-existed with global hyposexuality, which they defined as the individual not having at least one episode of intercourse or sexual arousal no matter what the stimulus every 2 months. Of six patients who had developed CPS between the ages of 13 and 15 years there appeared a near total lack of sexual response. Two of the patients were middle aged men both of whom had satisfactory sex lives, who became impotent within a year of developing temporal lobe tumours. After operation those who got relief from seizures were found to have an increase in sexual interest including one man who having developed CPS at age 12 experienced his first ejaculation age 30 and subsequently married. Two patients were said to have passed through a phase of hypersexuality post operation, although this is not defined, which was terminated by the return of seizures. Once again no mention is made of AED therapy. Cogen et. al. (1979) looked at 25 patients who underwent temporal lobectomy, and

found sexual dysfunction in 5 men and 2 women. Three of the men reported reduced libido, 1 increased libido and 1 had orgasmic sensations associated with seizures, a rare phenomenon according to Remillard (1983). The two women reported diminished libido. No mention is made of how this information was obtained, and increased libido or hypersexuality was defined as hetero or homosexual promiscuity, although this latter term is not defined. All patients had been treated with therapeutic doses of first line AEDs which were continued from three to six months after operation depending on seizure frequency. One man and two women reported improved sexual function postoperatively. Seizures were completely abolished by this surgery in some individuals and a 50 - 77% reduction in frequency was seen in the group as a whole. The authors, however, do not tell us exactly what happened to the seizure numbers in the patients who claimed an improvement in libido. The psychological effect of having the burden of frequent seizures removed, and the reduction in AEDs alone could account for the improvement. Jensen and Larsen (1979) investigated 74 patients with intractable CPS all of whom underwent temporal lobectomy. 63 of these patients, 37 men and 26 females were asked about sexual function. 19 males and 12 females reported sexual dysfunction most of them citing diminished libido as their principal problem. Their is no mention of the effect if any on libido of anterior temporal lobe resection, or of AED therapy.

Earlier papers by Falconer et. al. (1955), and Hill et. al. (1957) do mention in passing changes in sexual function in patients who had undergone temporal lobectomy. Both make the point that those who gained relief from seizures were most likely to report an

amelioration in their sexual interest and performance. Which begs the question of how much this improvement is due to social factors.

There have been a number of studies undertaken on patients living in the community for whom surgery was not considered suitable or whose seizure disorder was adequately controlled by AEDs. Saunders and Rawson (1970) studied 100 men with epilepsy all of whom resided in the community, 33 of whom suffered from CPS. Of these 33, 12 reported sexual dysfunction. 11 reported impotence, 4 impaired libido. All of the men had developed their seizures in their teenage years, but the authors do not tell us anything about the age at which they went through puberty. Of particular interest is the finding that the two men who complained of severe impairment of libido had both developed CPS in their early 50's and had satisfactory sexual relations within marriage for many years. All the patients were being treated with either phenytoin or a barbiturate at the time of the study. One patient in this study was found to be excessively drowsy on phenobarbitone and reported a significant improvement in sexual interest after changing to phenytoin. Saunders and Rawson wrote that they believed AED therapy to be a rare cause of impaired libido. Shukla et.al. (1979) investigated 70 cases each of CPS and primary generalised epilepsy. They found over 60% of the men and women in the CPS group were hyposexual. Hyposexuality was defined as less than one sexual outlet per month in the form of hetersexual intercourse. Deviation was defined as any form of sexual activity outwith "heterosexual genital union", occasional masturbation was allowed in the unmarried, and those not living with their partner. This study was performed in India, and it is unlikely that such definitions would not go

unchallenged in this country. However once again a pattern of global diminution of interest in sex appears particularly in those men who developed epilepsy before puberty, and a reduction in sexual activity since the onset of seizures. The women in this study did not receive quite the same degree of scrutiny as their male peers. We are told they took part in intercourse only on repeated requests by their husbands, remained "passive" and did not reach orgasm. Western feminism would infer from those statements that the problem lay with the husband not the wife! Shukla et al make no mention of these patients' AED regime, making it impossible to make any inference about the role of these drugs.

Pritchard (1980) studied 33 adult men with CPS and reported that 16 were hyposexual, although he does not tell us how he defined hyposexuality. 11 of the men were impotent, 3 had reduced libido, 2 suffered both. They found no difference in the age of onset of disease in those who were judged hyposexual as opposed to those who were not. Like Saunders and Rawson the men who had developed seizures after puberty and the older the man the more likely he was to report sexual dysfunction. They could find no correlation between drug level or seizure frequency and degree of impairment of sexual interest.

Fenwick et. al. (1985) studied 97 men all resident in a centre for those with severe epilepsy. 21% of these men had had sexual intercourse. When asked about orgasm whether achieved by intercourse or masturbation 44% said they had never experienced one. 20% claimed not to have reached orgasm in the last year. Interestingly 31% of the

sample said they had a moral repugnance about masturbation. 43% told the investigators that they had never had an orgasm during dreaming and 52% denied any sexual dreams.

One third of the sample reported difficulty in achieving and maintaining an erection, and 25% said they were anxious that sexual activity would precipitate a seizure. Fenwick could find no relationship between seizure type, and frequency. Neither was there any strong relationship between drug levels and sexual difficulty. This study was designed to inquire into aspects of male sexual function that are believed to be influenced by androgens, questions pertaining to spontaneous morning erections and daytime tumescence were included and a significant proportion, 35% and 48%, respectively reported never having experienced them. Again there was no convincing correlation with drug levels and these findings.

The same group then went on to compare three separate sets of men all of whom suffered from epilepsy (Toone et. al 1984). They were drawn from a residential home for people with epilepsy, a hospital epilepsy clinic, and general practise. Each man underwent a structured interview by a psychiatrist during which they were asked about their sexual lives. Four measures of sexual activity were recorded. Frequency of sexual activity resulting in orgasm, early morning erections, spontaneous daytime tumescence, and erectile impotence. They found 44% of these men claimed never to have has sexual activity that led to orgasm. 35% and 48% said they had never experienced spontaneous morning or daytime erections respectively. Similar inquiries were made of the men

attending the epilepsy clinic, and the another group being treated by general practitioners. Toone et al found that fewer of the men living in the community reported diminished libido, however, one third of these men were found to deny sexual thoughts, in stark contrast to 37 volunteers drawn from hospital staff who acted as a control group. Toone and colleagues could find no difference between those men suffering CPS in comparison to those suffering other forms of epilepsy.

Studies devoted exclusively to women are rare in the literature. Ndegwa et. al. (1986) studied a group of 30 women all in stable heterosexual relationships living in the community compared with a randomly selected group of women attending their general practitioner for non-sexual problems. There were no significant differences between the two groups in terms of age at time of study and length of relationship. Using the Griss questionnaire (Golombok Rust Inventory for Sexual Satisfaction; Rust et al 1986). The women with epilepsy were found to have intercourse less frequently than the control group, and to report vaginismus more frequently. There were no differences between the two groups of women in terms of achieving orgasm, dissatisfaction with their sexual relationship or of non-sensuality. No mention is made of AED regime or of seizure type and frequency. The authors concluded that their findings were compatible with reduced capacity for arousal rather than orgasmic dysfunction, in women with epilepsy.

Demerdash et. al. (1991) examined 327 women with epilepsy. They were divided into three groups. One group of women with epilepsy who were known to have a psychosexual disorder, a second group of randomly selected women with epilepsy, who

did not report any sexual difficulties, and a control group of healthy females recruited from a dental clinic. Because of its design no comment can be made on the true incidence of psychosexual dysfunction in this group of women. Of the 127 women in the group known to have psychosexual dysfunction inhibited sexual desire and sexual excitement were the two commonest complaints with 24% and 25% respectively of the women reporting these. A significantly greater number of those in the sexual dysfunction group suffered from CPS than those in the non-dysfunction group. In addition the women in this group were more likely to have abnormal EEGs, and to have significantly longer histories. Another point of interest in this group, which will be returned to later was the finding of a higher incidence of menstrual abnormalities in the sexual dysfunction group, compared with both the control and non-sexual dysfunction groups. There was no demonstrable differences between the two groups in type of AED therapy.

Demerdash reports that 11% of the sexual dysfunction group displayed exhibitionism, then goes on to describe this as occurring in the postictal period or as part of an automatism. A quite different phenomenon from the person- usually male -who knowingly exposes their genitals.

Jensen et. al. (1990) studied a group of 86 men and women with epilepsy all of whom were, like our study population, outpatients living independently in the community. This study is probably the best to date in the literature pertaining to sexual function and epilepsy in that a rigorous sexological approach was used using standard questions

aimed at exploring the extent of sexual desire as well as specific areas of dysfunction such as erectile failure, premature or retarded ejaculation, orgasmic dysfunction and vaginismus. The results were compared to earlier results obtained from a group of outpatients with diabetes and a non patient control group. There were no significant differences between

these three groups with the patients with epilepsy reporting similar levels of coitus and masturbation as the other two groups. Neither did the patients with seizures suffer from inhibited sexual desire or difficulty in achieving orgasm. This group also explored the degree of disease acceptance in patients studied and found a high degree of this suggesting that most of the individuals did not find their seizures a significant problem.

Jensen and his colleagues also measured hormone levels and found no significant differences in free testosterone between the patients taking AEDs and those who were not. Slightly reduced levels of free testosterone were recorded in 3 men but none of these men reported sexual difficulties.

Bergen et. al. (1992) reported on a group of randomly selected women attending an outpatient epilepsy clinic, using a group of women of similar background and mean age as a control. The women were asked to respond to twenty questions relating to sexual activity and libido. They found no significant relationship between sexual activity and desire, duration of epilepsy, seizure type, age or type of AED regime. They found a bimodal distribution of answers. One group of women with seizures appeared to exhibit sexual desire and intercourse, although the number of occasions they reported this to

happen tended to be less, but not significantly so, compared to the control group. A second, but smaller group of women appeared distinctly hyposexual with some reporting never experiencing sexual desire or intercourse. Because some of the women with epilepsy had no regular sexual partner their responses were eliminated after the first

analysis and the data from those women with partners looked at again. The same phenomenon was observed. There seems to be no immediately obvious explanation for this observation. Bergen et al do not mention anything of the social circumstances of the patients or of their partner's disposition. And there were no differences in AED therapy. AED levels were not ascertained in this study. There is, therefore evidence to suggest that some people with epilepsy suffer from hyposexuality, although no agreement as to its causes.

POSSIBLE MECHANISMS OF HYPOSEXUALITY

TEMPORAL LOBE PATHOLOGY.

Mesial temporal sclerosis with loss of neurones in the hippocampus and amygdala is the commonest finding in people undergoing temporal lobe resection for intractable CPS (Falconer et. al. 1955; Falconer et. al. 1964; Falconer et.al. 1969; Bruton 1988; Rausch 1991). These structures appear to be of fundamental importance in motivational behaviour such as feeding reproduction and social interaction in both non-human primates and lower mammals (Gloor 1972).

In 1939 Kluver and Bucy performed bilateral temporal lobectomy on a series of rhesus monkeys, and observed a profound change in these animals cage bound behaviour. They developed " psychic blindness " or visual agnosia, approaching objects and individuals from which they had previously fled. They ceased to display the vocal and motor reactions usually associated with fear and anger, and showed a marked tendency to investigate every object encountered orally. In addition there was a striking increase in sexual activity with animals making indiscriminate attempts at copulation. None of these changes were seen when unilateral temporal lobectomy was performed. Kluver and Bucy noted that these behavioural changes occurred when the uncus and the greater part of the hippocampus and amygdala were removed bilaterally. Resection of the first, second and third temporal convolutions, and severing of the fronto - temporal and occipital - temporal pathways did not induce the syndrome

Monkeys with bilateral amygdalar lesions rather than complete temporal lobectomies exhibited the same change in behaviour (Akert et.al. 1961; Dicks et. al. 1969; Kling

1972) But the changes in sexual activity originally described by Kluver and Bucy seemed to occur only when the animals were caged, especially if confined with one other animal who had undergone similar surgery. Of particular interest, was the observation that when these monkeys were released back into the wild they tended to become social isolates, ceasing to indulge in mutual grooming, the staple social interaction in primate society, and loosing their position in the social hierarchy (Kling 1968; Dicks et. al. 1969; Kling 1972). This loss in rank reduced the males' access to females. In addition the operated animals displayed fear in the face of friendly approach by other monkeys, and in the case of one female a very marked disinclination to indulge in social foreplay prior to mating with an unoperated male (Dicks et. al. 1969).

Effects similar to those seen in the Kluver Bucy monkeys have been reported in humans. In 1955 Terzian and Ore described the case of a 19 year old youth who underwent bilateral temporal lobectomy for intractable seizures. Post operatively the boy exhibited a flat affect, voracious appetite, impaired memory, made persistent sexual overtures to male members of staff , which were easily rebuffed, and masturbated openly. He failed to recognise people even close relatives and did not display any form of emotion towards them, despite having a warm and affectionate relationship with his mother before the operation. His condition remained unchanged at follow-up two years latter. Lilly et. al. (1983) reported 12 cases of Kluver-Bucy syndrome caused by herpes encephalitis, Picks, Alzheimers, and trauma. All of these patients displayed some if not all of the features delineated by Terzian and Ore. They found that hypersexuality and sensory

agnosias were the components of the syndrome least frequently seen, placidity and exaggerated oral activities occurred most often in partial syndromes.

Unilateral damage by contrast appears to cause a diminution in sexual activity as already described above. Hierons et. al. (1966) reported the cases of 15 men 8 of whom had neoplastic or traumatic lesions of one temporal lobe and reported that although libido was retained the men became impotent.

Visual agnosia is a feature of Kluver Bucy syndrome and was present in the boy studied by Terzian and Ore. Visual perception of objects and their significance is a function of the inferior temporal neocortex (Kluver et. al. 1939; Gloor 1972), which has connections with the amygdala and hippocampus (Gloor 1972). Motivational behaviour in man and non-human primates is guided by visual stimuli (Gloor 1972) and any interruption in their processing at amygdalar level could compromise the individuals capacity for social interaction, as has been demonstrated in monkeys (Kling 1968; Dicks 1969; Kling 1972)

The amygdala can be divided into two discrete parts. Corticomedial which regulates those hormones concerned with homeostasis (Zolovick 1972; Dreifuss et. al. 1986; Herzog 1986), and basolateral which exerts actions on reproductive functions (Zolovick 1972; Dreifuss et. al. 1986; Herzog 1989). Destruction of the basolateral part of the

amygdala in adult female deermice causes anovulation and cystic changes in the ovaries, in addition to changes in serum LH and FSH in both sexes of these mice (Zolovick

1972). Amydalectomy in adult male rats causes atrophy of the testes (Yamada et. al. 1960), lesions placed in the basolateral area of the amygdala in cats causes hypersexuality after a latent period of six to eight weeks (Wood 1958), a phenomenon observed in certain humans after temporal lobectomy (Blumer 1970). Bilateral temporal lobectomy in female monkeys induces long periods of amenorrhoea (Wada et. al. 1961), although unilateral lesions do not. The reason for these findings is believed to lie in the finding that the amygdala has extensive direct anatomical connections with the ventromedial and pre-optic nuclei of the hypothalamus (Renaud 1976), enabling it to influence gonadotrophin concentrations in both the pituitary and the blood.

Although our understanding of the reproductive functions of the amygdala comes from non-human studies there is a body of evidence that men and women with complex partial seizures sustain a higher than expected incidence of reproductive endocrine disorders, which cannot be explained by AED therapy. Hypogonadol hypogonadism, polycystic ovarian syndrome, and oligomenorrhoea have been reported to occur more frequently in these patients than in the population as a whole (Herzog et. al. 1982; Herzog et. al. 1986; Herzog et. al. 1989). These patients have also been noted to produce fewer offspring than their peers (Lennox et. al. 1953; Webber et. al. 1986). In addition those who develop seizures in the first decade of life are less likely to marry and produce offspring than those who had their first seizure in the second decade of life

(Lennox et. al.1953; Lindsay et. al. 1979). The association between hyposexuality and early age at onset of seizures may say more about the importance of the type of epilepsy the individual suffers from rather than the number of fits sustained. French and colleagues (1993) studied 67 patients all of whom had been rendered seizure free by temporal lobectomy, the majority of whom were found to have mesial temporal sclerosis at operation. There was a strong association between the occurrence of complicated febrile seizures and the latter development of CPS. There was a mean interval between the time of the initial insult to the onset of habitual seizures of 7.5 years, implying that the majority of patients in their study developed CPS in the first decade of life. It was also noted that as time went on some patients reported their auras became more elaborate suggesting a progression of the initial lesion. Volumetric measurements of the hippocampus and amygdala using magnetic resonance imaging (MRI) in cases of CPS have shown significant atrophy of these structures, and a high level of agreement between the side on which atrophy has occurred and EEG lateralisation (Cendes et al 1993,a). Further studies by the same group failed to show any significant correlation between estimation of seizure frequency, duration of epilepsy or age of the patient and the degree of hippocampal / amygdalar atrophy. However patients with a history of prolonged febrile convulsion had significantly reduced amygdalar and hippocampal volumes in comparison to those who had not (Cendes et. al. 1993 b). Whether atrophy of the mesial structures is a cause or consequence of seizures remains unresolved. The temporal lobes in man contain areas involved in visual, and auditory perception, including language. Closely allied to these areas are the amygdala and hippocampus which in turn are linked to the hypothalamus which mediates basic drive mechanisms

(Gloor 1972). Damage to the amygdala and hippocampus at an early stage in life may prevent the integration of those higher functions such as language which are situated in the neocortex with basic drives leaving the affected individual unable to recognise, or unmoved by, social cues employed by the opposite sex to encourage intimacy.

DESCRIPTION OF ANTIEPILEPTIC DRUGS

Phenytoin (PHT), Sodium Valproate (VPA), and Carbamazepine (CBZ), have been first line AEDs for the last 20 years, and it appears likely that they will remain so for the foreseeable future. Three new AEDs, Vigabatrin, Lamotrigine, and Gabapentin have made their therapeutic debuts in the early 1990's, but in the United Kingdom only Lamotrigine has been licensed for use as monotherapy. All of the patients who participated in this study were receiving one or other of these first line agents either alone or in combination with one of the others. None were receiving phenobarbitone or one of the recently introduced compounds.

SODIUM VALPROATE

VPA first became available for use in the United Kingdom in 1974 (Lancet 1988) and although metabolised in the liver is the only one of the major AEDs not to induce hepatic monoxygenase enzymes (Brodie et. al. 1988). It can inhibit drug oxidation increasing circulating concentrations of PHT and CBZ, and is thought to act by enhancing gamma - amino - butyric - acid (GABA) levels in brain (Lancet 1988).

VPA is indicated for the treatment of the primary generalised epilepsies (Wilder et. al. 1983 ; Callaghan et. al .1982) and myoclonic epilepsy (Clement et. al. 1988). Its use in partial epilepsy is less certain (Lancet 1988). Like other AEDs it has its share of side effects (table 1, page 28). There is little correlation between VPA concentration and pharmacological effect (Minns et. al. 1982).

PHENYTOIN

Phenytoin was introduced in 1938 (Merrit et. al. 1938) and is effective against both generalised tonic - clonic and partial seizures. PHT modifies the tonic phase in the maximal electric shock test, diminishing after discharge generation and preventing the spread of seizure discharge (Woodbury 1980). In addition PHT acts as a membrane stabiliser by enhancing sodium extrusion from cells and inhibiting passive sodium entry (Woodbury 1980). PHT inhibits calcium from entering depolarised synaptosomes thus diminishing the release of neurotransmitters (Woodbury 1980), and is thought to modulate the GABA receptor (Eadie 1984).

PHT is metabolised in the liver by hydroxylation followed by glucuronidation (Eadie 1984), and exhibits saturation kinetics, thus at higher doses a further small increase in dose can result in a disproportionate rise in serum levels (Eadie 1984). When administered to people taking carbamazepine PHT can cause a decline in CBZ levels. Women using the oral contraceptive pill require a " pill " with a high oestrogen content because of increased hepatic metabolism induced by PHT (Mc Innes et. al. 1988). Unlike VPA there is a relationship between PHT levels and pharmacological effect (Lund 1974).

PHT has a large number of side effects (Table1, page 28). In recent years there has been an increasing disinclination to prescribe the drug particularly in young women because of its cosmetic side effects, and interaction with the oral contraceptive pill. Nonetheless it remains an important AED.

CARBAMAZEPINE

Carbamazepine was introduced in the early 1960's and is effective in the treatment of simple partial, complex partial, partial with secondary generalisation and tonic - clonic seizures (Leppik 1990), like PHT it is not effective against absence attacks (Leppik 1990). It is metabolised in the liver with the formation of carbamazepine epoxide and induces its own metabolism thus requiring modification of dosage several weeks after its introduction (Faigle et. al. 1982). Because CBZ is an enzyme inducer when used in combination with PHT the two drugs can mutually induce or inhibit elimination thus causing higher or lower than expected levels of both drugs. CBZ can increase clearance of VPA, but VPA can reduce protein binding of CBZ and inhibit CBZ breakdown. As with PHT the clearance of exogenous steroids is increased in people receiving CBZ, women using the oral contraceptive pill should be advised that they require a preparation with increased oestrogen content.CBZ acts on sodium channals, reducing sodium currents and thus stabilising membranes (Mc Lean et. al.1986). Like PHT and VPA it has it's share of side effects (table 1, page 28), and in common with PHT there is a correlation between therapeutic effect and blood level (Leppik 1990)

Table 1: Side Effects of Established Antiepileptic Drugs

CARBAMAZEPINE	PHENYTOIN	SODIUM VALPROATE
*Dizziness	*Ataxia	*Tremour
*Headace	Anorexia	*Hair fall
*Nausea	Dyspepsia	Anorexia
Drowsiness	Nausea	Weight gain
Neutropenia	Vomiting	Dyspepsia
Hypocalcaemia	Aggression	Nausea
Hyponatraemia	Depression	Vomiting
Oralfacial dyskinesia	Drowsiness	Alopecia
Cardiac arrythmia	Headache	Peripheral oedema
	Paradoxical Seizures	Drowsiness
	Megaloblastic anaemia	Hyperammonaemia
	Hyperglycaemia	
	Hypocalcaemia	
	Osteomalacia	
	Neonatal Haemorrhage	
*Morbilliform rash	*Acne	Acute pancreatitis
Agranulocytosis	*Gum hypertrophy	Hepatotoxicity
Aplastic anaemia	*Course Facies	Thrombocytopenia
Drug induced lupus	*Hirsutism	Stupor
Hepatoxicity	Blood Dyscrasias	Encephalopathy
Photosensitivity	Lupus like syndrome	Teratogenicity
Teratogenicity	Reduced serum IgA	
Stevens Johnson Syndrome	Pseudolymphoma	
Thrombocytopenia	Peripheral Neuropathy	
	Rash	
	Stevens Johnson Syndrome	
	Dupytrons contracture	
	Hepatotoxicity	
	Teratogenicity	
Above Line: Predictable	**Below Line: Idiosyncratic**	*** Common Problems**

ANTIEPILEPTIC DRUG THERAPY

Antiepileptic Drugs (AED) were not considered a contributory factor in the early reports of hyposexuality. Von Krafft - Ebbing noted " indifference " in 1892, whilst Gastaut and Collomb (1954) and Taylor (1969) published their reports on the phenomenon before carbamazepine and sodium valproate had been added to the roster of anticonvulsants.

In 1975 Christiansen and colleagues reported diminished urinary excretion of aldosterone and dihyroepiandosterone sulphate in male patients receiving AEDs. In 1977 raised sex hormone binding globulin (SHBG) was reported in women taking phenytoin (Victor et. al. 1977), and the following year raised SHBG was reported in a group of men and women receiving several different AEDs (Barragry et. al. 1978). This same study noted that there was no increase in Vitamin D binding globulin, T3 resin binding or thyroxine, implying a selective effect on hepatic SHBG synthesis by AEDs. None of the men or women in this study reported sexual difficulties.

Since then numerous studies have revealed increased SHBG in men and women taking AEDs (Backstrom et. al. 1979; Toone et. al. 1980; Dana - Haeri et. al. 1982; MacPhee et. al. 1988 ; Isojarvi et. al. 1990 ; 1991: Jensen et al 1990).

At the same time changes in serum androgen levels were noted. Raised total testosterone in men taking AEDs was reported (Barragry et. al. 1978; Toone et. al. 1980). In 1982

low free testosterone levels in male patients were first reported (Dana - Haeri et. al. 1982; Toone et.al.1982), and the following year Toone et. al. (1983) studied 129 men in the David Lewis Epilepsy centre finding significantly reduced free testosterone and total testosterone in comparison to a control group of men drawn from hospital staff. Subsequent studies also recorded reduced free testosterone (Toone et. al. 1984; 1986 McPhee et. al. 1988)

Other studies, however, failed to demonstrate a drop in free testosterone. Backstrom et. al. (1979) studied six women all of whom were taking phenytoin and found calculated free testosterone did not differ significantly from a control group although the free androgen index of the treated women was lower than the control group. Rodin et. al. (1984) studied 33 men and found total testosterone levels within the normal range. Isojarvi et. al. (1988) could find no significant differences between in total or free testosterone between a group of men receiving carbamazepine monotherapy, a healthy control group, and a group of untreated patients. In a further study (1990) the same group examined the effects of carbamazepine monotherapy on a group of female patients, and found them to have a significantly reduced free androgen index in comparison with the control group , but no significant difference in free testosterone

levels between the groups. Jensen et. al. (1990) found no significant differences in free testosterone levels in men and women taking AEDs compared with controls.

There appears to be universal agreement in the literature that enzyme inducing AEDs either alone or in combination are associated with a rise in SHBG (Victor et. al. 1977; Barragry et. al. 1978;Backstrom et. al. 1979;Toone et. al. 1980;Connell et. al. 1984; Mac Phee et. al. 1988;Isojarvi et. al. 1988;1991). The mechanism underlying this change is not understood. Induction of the hepatic monoxygenase enzyme system leading to increased disposal of testosterone causing a fall in serum levels which would stimulate SHBG levels (Anderson 1974) has been suggested. Alternatively enzyme inducing AEDs may stimulate SHBG synthesis by the liver. The increased testosterone binding capacity causing an increase in total testosterone, but a fall in free testosterone which would in turn cause a further rise in SHBG production. The antituberculous agent rifampicin, like phenytoin and carbamazepine, induces the hepatic monoxygenase system, and its ingestion is associated with a rise in SHBG (Brodie et. al. 1981). Connell et. al. (1984) found that administration of carbamazepine to healthy male volunteers was followed by an acute fall in total testosterone, free testosterone fraction androstenedione and DHEAS, and a rise in SHBG levels. By the end of the 21 day study total testosterone, free testosterone fraction and androstenedione had all returned to baseline levels. Connell concluded the changes were due to carbamazepine induction of SHBG synthesis and the hepatic monoxygenase system. Each of these changes tending to reduce free testosterone levels. The return to baseline of the free testosterone

fraction, total testosterone and androstenedione was thought to be due increased testicular steroidogenesis, although there was no demonstrable increase in LH levels.

Studies aimed at examing the relationship between individual AEDs and hormone levels have been inconclusive. Using simple linear regression Toone et. al. (1980) found positive correlations between phenytoin levels and total testosterone. In 1983 the same group reported positive correlations between carbamazepine and prolactin levels. They also found positive correlations between sodium valproate concentrations and both total testosterone and LH levels none exceeded $r = 0.53$, however, and there was no demonstrable relationship between the sexual behaviour of the men studied and their AED levels. Rodin et. al. (1984) found a significant correlation between carbamazepine and prolactin ($r = 0.63$) and Mac Phee et. al. (1988) noted a negative correlation between carbamazepine levels and calculated free testosterone ($r = - 0.54$). In contrast neither Barragry et. al. (1978) or Isojarvi et. al. (1988;1991) reported significant relationships between AED concentrations and hormone levels.

The effects of AEDs on the central mechanisms controlling gonadotrophin and prolactin secretion have also been explored. Gallagher et. al. (1984) studied secretion rates of cortisol and ACTH in a group of five patients with CPS treated with AEDs, comparing them to five patients with none epileptic seizures (NES) who were also taking AEDs. The patients with CPS had significantly higher rates of ACTH and cortisol secretion than the NES group. In the next part of this study ACTH and cortisol was measured in 9 healthy controls, and compared with seven patients suffering CPS treated with AEDs

and 11 patients treated by temporal lobectomy. Surgery restored rates of secretion to normal whether seizures were fully abolished or not. As the surgically treated patients continued to take AED therapy the authors concluded that these drugs were not responsible for the observed changes and suggested the disruption of pathways linking the medial temporal lobe to the hypothalamus reduced secretory rates. In contrast Francheschi et. al. (1984) could find no differences in ACTH or cortisol secretion in a group of 63 men and

women all treated with AEDs when compared to a control group of healthy volunteers. Stimulation of plasma growth hormone (GH) with L-Dopa showed those treated with sodium valproate alone had significantly lower peak levels, whilst those taking phenytoin or multiple AED attained significantly higher peaks than the control group. A similar pattern to this was seen when metoclopramide was administered and peak prolactin levels measured. Those men taking sodium valproate did not differ from the control group in their response to the stimulus, however, those men taking phenytoin, phenobarbitone, carbamazepine or a combination of these drugs exhibited significantly higher mean peak values than the control group. There was no difference between the women patients and control group in prolactin responses. In this study basal gonodotrophin levels did not appear to be affected by AED therapy. Neither did seizure type, duration of epilepsy or age seem to affect any of the parameters studied. Further studies have shown abnormal responses to dynamic pituitary testing. Dana - Haeri et. al. (1984) studied 19 patients taking carbamazepine or phenytoin. Basal LH was significantly raised in the male patients with an exaggerated rise in LH in response to LH- RH TRH administration in those men taking carbamazepine. Two hours after

injection of LH -RH / TRH prolactin levels were significantly higher in the women taking AEDs than in the women in the control group. Herzog et. al. (1986 a) examined 50 consecutive women with CPS 30 of whom took AEDs 20 of whom did not. 28 of these women reported irregular menstruation, 19 exhibited gonadotrophin and ovarian changes in keeping with polycystic ovarian disease in 10 cases and hypothalamic hypogonadism in 9. There was no significant difference between the treated and untreated groups in the incidence of these conditions, and no demonstrable relationship between any particular AED and menstrual

dysfunction. Furthermore the authors demonstrated that those women with polycystic ovarian disease were more likely to have left sided EEG foci, those with hypothalamic hypogonadism a right sided focus. The same group then investigated 20 consecutive men with CPS reporting that 11 of them suffered a reduction in sexual interest or potency. 5 of these men had low LH and testosterone levels, and subnormal maximum responses to LH - RH. Two had persistently raised prolactin levels, and a further two raised LH levels but abnormally low free testosterone levels. Once again Herzog makes the point that 3 of the men deemed to have hypothalamic hypogonadism had not been treated with AEDs. In an earlier paper (Herzog et. al. 1982) looked at 4 men and 3 women none of whom was receiving AEDs. The men had significantly lower basal LH than the control group, and both men and women exhibited a wide variation in LH response to LH - RH from the sub to the supra-normal. Other studies have failed to demonstrate any connection between AEDs and gonadotrophin levels or responses. Isojarvi et. al.(1988) investigated 23 men receiving carbamazepine monotherapy, 18 untreated men, and 19 age matched controls finding no difference in gonadotrophin

levels between the groups. No dynamic pituitary testing was performed. The same group then studied a group of 40 men and women taking carbamazepine only comparing them with a group of 29 untreated patients. Unfortunately details of seizure type are not provided in either of these papers, but the authors state that most of the patients had been seizure free for a year prior to the study. Mean basal LH was significantly lower in the carbamazepine treated women than in the untreated females, with blunting of LH response to LH-RH in the treated women. There was no difference in basal or stimulated FSH levels. Mean basal prolactin levels were significantly lower in the treated

men, with significantly greater maximal prolactin response to TRH. All the women were noted to have regular menstrual cycles. A prospective study of 13 women commenced on carbamazepine showed that basal LH levels fell within the first year, and that LH response to LH-RH was significantly diminished after two months of drug therapy, FSH and prolactin levels remained unchanged (Isojarvi et. al. 1990). In contrast Isojarvi and colleagues reported decreased basal LH with an increased LH - RH response in men receiving phenytoin monotherapy and a combination of phenytoin and carbamazepine. This same study found increased FSH response in a group of men taking both carbamazepine and sodium valproate, but reduced basal FSH in men taking sodium valproate only (Isojarvi et.al. 1990).

Mac Phee et. al.(1988) noted raised basal LH in men treated with carbamazepine monotherapy, and also in those taking more than one first line AED. There was no significant differences in gonadotrophin response to LH-RH in men taking phenytoin,

sodium valproate or carbamazepine, singly or in combination. Rodin (1987) showed an inverse relationship between age and FT levels suggesting that there was a premature ageing of the reproductive system in men with epilepsy. Increased basal LH levels have been reported in men taking AEDs (Toone et. al .1980; Dana - Haeri et. al. 1984; Herzog et. al. 1986; Mc Phee et. al. 1988). A raised LH is in keeping with a reduced FAI or FT (Johnson et. al. 1991, page 126). The fact that FT levels do not seem to return to normal under LH drive in the studies cited above has led to the proposal of a direct toxic effect on testes by AEDs (Toone 1986), compromising their ability to

respond to pituitary hormones. In addition AEDs have been reported to reduce sperm motility (Chen et. al. 1992) and cause abnormal sperm morphology (Christiansen et. al. 1975). Thus men taking AEDs may suffer a two pronged attack on their reproductive system.

The majority of studies of gonadotrophin response have been performed on patients with CPS. Bilo et. al. (1988) studied a group of 20 women all of whom suffered from PGE. Drug therapy consisted of sodium valproate and phenobarbitone in combination,. or as monotherapy, three of the women were not receiving any AED. Six of these twenty women complained of long-standing menstrual irregularity, of these 3 were found to have polycystic ovarian disease, and two hypothalamic ovarian failure. One of the six women did not take any AED, and three of these women had developed menstrual irregularity before the inception of drug therapy. The fourteen women with

regular menstrual cycles had higher mean basal prolactin and FSH levels than the control group, and significant blunting of LH response to LH - RH.

In conclusion there is no firm consensus on the effects if any of AEDs on the pituitary - hypothalamic axis. Individual research groups achieve a certain consistency in their reports, but more prospective studies of the type undertaken by Isojarvi in 1990 are needed.

Raised prolactin levels are frequently reported after tonic - clonic and CPS (Trimble et. al. 1978; Pritchard et. al. 1983), an observation used as an aid to distinguish between NES and epileptic seizures, and amygdalar stimulation in the rat is known to result in release of gonadotrophins (Velasco et. al. 1968). In humans amygdalar stimulation has been reported to cause elevation in prolactin by some (Parra et. al. 1980), but not by others (Sperling et. al.1986). Endocrine function following CPS without secondary generalisation in men where studied by Pritchard et. al. (1983). All of these men were taking at least one AED, and all had interictal gonadotrophin, prolactin, cortisol and GH levels within normal range. A significant but transient rise in prolactin was noted postically, but not in the other hormones studied. Aminoff et. al. (1984) studied 20 patients with generalised tonic-clonic attacks finding significant elevation in prolactin, ACTH, cortisol and vasopressin postically, but no change in gonadotrophin levels. These changes did not persist for more than 2 to 3 hours at most. Thus the evidence that the changes in endocrine function that some patients with epilepsy sustain are consequent upon subclinical seizure

activity in the limbic system is not compelling, and the levels of prolactin observed are not of the same order as those seen in prolactinomas (Franks et. al. 1978) making it unlikely that sexual indifference and impotence in men with epilepsy are caused by postictal hyperprolactinaemia.

AEDs are known to affect cognition (Trimble 1987), with phenytoin in particular being associated with significant cognitive impairment (Trimble 1987). Although it is acknowledged that disentangling the underlying disease process from drug side effects can be difficult. Gillham et. al.(1988) administered a battery of psychometric tests to 85 patients with epilepsy 26 of whom were untreated, 40 received carbamazepine monotherapy and 19 took CBZ along with another AED. They found that the polypharmacy group reported greater sedation than the other two groups and performed less well on choice reaction time, paired association learning and back digit time. The only significant difference between the carbamazepine and control group was in movement detection on a video screen. In a further study, Gillham et.al. (1990) 110 patients were studied this time using a healthy control group as well as a group of people with epilepsy not receiving AEDs. There were no significant differences between the healthy controls and untreated group. Carbamazepine treated individuals scored less well on psychomotor tests than the sodium valproate, control and untreated groups and phenytoin had poorer composite memory scale scores than the other groups. Pulliainen et.al.(1995) studied 31 patients randomly assigned to either carbamazepine or phenytoin monotherapy over a two year follow up period and found after the initial few months that the only difference between the two groups was was phenytoin treated

patients has a lower visual memory score than the carbamazepine group. There was, however, no control group in this study. Thus the sedative and possible cognitive side effects of AEDs may also contribute to a lack of sexual interest.

PSYCHOSOCIAL EFFECTS OF EPILEPSY

There is an extensive literature on this subject, and an exhaustive review of it is beyond the scope of this work. In the nineteenth century the French physician Billod (1843) summed up the lot of the " epileptic " thus.

'Usually the epileptic is avoided:on all faces he reads his sentence to isolation.Everywhere he goes menacing and insurmountable obstacles arise to his obtaining a position,to his establishing himself,to his relationships and to his very livelihood: he has to say goodbye to his dreams of success for the masters even refuse him work in shops;goodbye to his dreams of marriage and fatherhood, goodbye to the joys of the domestic hearth. This is death to the spirit.'

One of the problems in this area of research is sorting out which psychosocial effects are due to a chronic and disruptive condition and which are due the epilepsy. Asthma and diabetes are sometimes cited as comparable chronic conditions, yet neither of these illnesses carries the stigma that is still attached to epilepsy. Epilepsy is regarded as unique in its capacity for causing social embarrassment. The sufferer is overwhelmed by a sudden apparently extraneous force which renders them incapable of rational behaviour One, albeit rare condition which does approach epilepsy in its potential for social embarrassment is narcolepsy / cataplexy. One study (Broughton et. al. 1984) looked at three groups comprising 33 women and 27 men. One group was comprised of healthy individuals, a second who suffered from narcolepsy / cataplexy, and a third

consisting of men and women with CPS and PGE, none of which had evidence of intracerebral pathology. They received PHT, CBZ, VPA, PB, either as monotherapy or in combination. In structured interviews those men and women with epilepsy suffered less social disruption on account of their seizure disorder, and seemed to have fewer employment problems than the narcolepsy / cataplexy group. The men in the epilepsy group had a higher incidence of impotence than the men in the control group, but this did not differ from the narcolepsy / cataplexy group. The paper does not state whether any further questions were asked to try and elucidate the cause of these mens' erectile failure. There were no differences between the women in the epilepsy and cataplexy / narcolepsy groups in the frequency with which symtoms were believed to increase in the premenstrual period. No inquiries were made of the women concerning sexual problems. Given the differences in drug therapy and, as far as we know pathological substrate, it could be argued that these effects reflected the men's self esteem, and anxieties about their illness and its potential for social embarrassment.

Adults developing epilepsy face numerous problems, not least of which can be unwitting prejudice on the part of the public at large. Hansson et.al. (1976) asked a group of American college students to examine a series of photographs of individuals of the opposite sex, and to indentify from purely facial charicteristics whom they considered to be suffering from epilepsy. Students of both sexes consistently chose a physically unattractive person. When challenged about his choice one student replied that he had worked with " epileptics " and he could identify them by the look in their eyes. Although such statements may seem at first glance risible there is a body of medical

literature purporting to describe the " epileptic personality ". Usually ascribed to individuals with CPS it comprises a constellation of unattractive traits. They are said to be " viscous " - a term much used but never defined (Small et.al. 1962; Bear et.al. 1977; Bear et.al.1982). Meticulous in attention to minor and often irrelevant detail (Waxman et.al. 1974; 1975), excessively religious and prone to aggression and psychosis (Bear et.al.1977; Flor - Henry 1976; Dewhurst et.al 1977). Tizard (1962) argued against its existance making the obvious point that the severely afflicted individuals who required close supervision and who most frequented the pages of these reports were a different population to those who lived and worked in the community.

A seizure disorder may simply make it more difficult to get work because of the belief that the individual concerned will require special consideration or simply have more sick time than a healthy person. A belief shared by some members of the medical profession (Beran et. al. 1981). Loss of a driving licence compounds this as well as curtailing freedom of movement. Women have the added burden of knowledge that AEDs have been implicated in fetal abnormalities (Yerby 1991), and that pregnancy can be associated with increased seizure frequency (So et. al. 1981), to say nothing of the fear of seizure whilst nursing or bathing a child. This combination of loss or limitation of job prospects, the physical effects of seizures, and the sedation and memory impairment reported by some adults commenced on AEDs could easily explain at least a degree of sexual indifference.

If the seeds of our future emotional development are sewn in childhood there is ample evidence pointing to the deleterious effect epilepsy can have on the young and their families. In 1972 Hartlage et al compared dependency rates in three sets of children under 12 years. One group suffered form seizures, a second from cystic fibrosis and the third an essentially healthy group undergoing routine tonsillectomy. No significant differences in parental attitudes were found between the three groups, but the children with epilepsy exhibited a higher degree of emotional dependency as assessed by validated questionnaires, then the children in the other two groups. Seizure type and AED therapy had no effect on these results. Hartlage noted that there was a tendency amongst the parents of children with epilepsy to try and suppress expressions of anger, aggression and sexuality by the affected child. Long et. al. (1979) interviewed a large number of parents of children with epilepsy, finding that the majority expected the child to underachieve at school, play fewer sports, be more highly strung and enjoy a limited choice of occupation in adulthood in comparison with their other children. Lechtenburg (1984) noted that in families with a child with epilepsy the mother appeared to be dominant, with her decisions on what activities were safe for the child overruling those of her husband's. In addition he described different strategies adopted by different families to cope with the problem. In some the mother encouraged emotional dependency in the child, in others there was a blatant denial of the existence of seizures effectively preventing the child and its brothers and sisters from articulating their fears or curiosity about the condition.

Matthews et. al. (1982) studied 15 children with seizures comparing them with age matched healthy controls and children with diabetes. The children with seizures, perhaps not surprisingly attributed control over their lives to outside agencies- parents, teachers God - and considered any successes they achieved to be due this external force.

They had lower self esteem, some reporting they were last to be picked for team games, and exhibited greater anxiety about everyday social situations. Children with epilepsy have usually been seen as prone to antisocial behaviour, and violence (Tizard 1962). More recent work, however, suggests that children are more anxious, fearful, and have fewer friends than their peers (Stores 1978; Hoare 1984:1991). Stores (1978) noted that it was boys with seizures who were more likely to exhibit these traits, along with a greater need for reassurance, often in the form of their mother, and physical affection, than were girls with epilepsy. In contrast Hoare (1991) found the same level of dependency in both sexes. Thus the social and psychological effects of epilepsy in an individual can be profound, and emotionally disabling. To say nothing of the effects of AEDs or temporal lobe dysfunction on reproductive mechanisms. What follows is an attempt to disentangle some of these factors.

SEX HORMONES

Sex steroids are all derived from cholesterol. Like their ubiquitous precursor they are lipid soluble but poorly water soluble circulating in the plasma bound to sex hormone binding globulin (SHBG), and albumin.

The first step in the formation of the sex steroids is the conversion of cholesterol to pregnenolone, a rate limiting step which takes place in the inner membrane of the mitochondria. From pregnenolone are formed the progestagens, oestrogens and androgens. Each of which makes a distinct contribution to human reproductive function.(See table 2, page 46)

Table 2:Actions of Sex Steroids

PROGESTAGENS	ANDROGENS	OESTROGENS
Prepare uterus for implantation	Induce and maintain differentiation of male somatic tissue	Stimulate secondary sexual characteristics in women
Maintain uterus during pregnancy	Induce secondary sexual characteristics in males	Prepare uterus for spermatozoal transport
Stimulates growth of breasts	Support spermatogenesis	Increase vascular permeability and tissue oedema
General mild catabolic effect	Influence sexual and aggressive behaviour in both sexes	stimulate growth and activity of breast and endometrium
Regulate secretion of gonadotrophins	Promote protein anabolism	mildly anabolic
	Regulate gonadotrophin secretion	Regulate gonadotrophin secretion

THE ANDROGENS

The androgens are a group of C 19 - steroids comprising testosterone, androstenedione (AND), dihydrotestosterone, dehydroepiandosterone, and dehydroepiandosterone sulphate (DHEAS). These steroids induce and maintain the differentiation of male somatic tissue, induce secondary sexual characteristics in men, and body hair in women, enable spermatogenesis to proceed, and in both sexes promote sexual interest or libido

Testosterone is the most abundant androgen in men. It is secreted at a rate of 24 μmol/day by the testes (Johnson et. al. 1991) and 80 % of this testosterone circulates bound to SHBG (Anderson 1974) The remainder being bound to albumin and cortisol binding protein. 2 % remains none - protein bound or free. In women androstenedione is the most abundant androgen in the blood. 50% coming from the adrenal cortex and 50 % synthesised in the ovary. Androstenedione is then converted to testosterone and dihydrotestosterone in the tissues (Bancroft 1989, p 27), providing half of the circulating testosterone, with the rest coming in equal parts from the ovary and adrenal 0.02 μ mol / day. 60 % of testosterone in women is bound to SHBG the rest to albumin and cortisol binding protein. 1 % remains free. Androstenedione and total testosterone levels are known to vary predictably with significantly higher levels in the mid - third of the menstrual cycle around the time of the LH surge, but with no significant differences in levels between the first and last third of the cycle (Judd et. al. 1973; Goebelsmann et. al. 1974).

Most workers accept that it is this free testosterone which is physiologically active, although receptors for SHBG have been identified in the uterus and prostate (Selby 1990) At the time when most of the studies of the effects of AEDs on sexual function and on testosterone levels were undertaken the methods available for measurement of free testosterone (FT) were time consuming, and too labour intensive for the use as a routine diagnostic test. Thus two methods were and are routinely used to estimate FT. One is a the free androgen index (FAI), a simple ratio of TT divided by sex hormone binding globulin (SHBG), the other an equation determined by Nanjee and Wheeler (1985) who studied 50 hirsute women using this ratio, and found 34 had a raised FAI implying increased FT. They also determined these women's' FT by direct measurement, and by mathematical formula which they had developed (see methods section for formula) finding that 34 and 37 of the women respectively had raised FT. They concluded that their equation was an adequate way of assessing androgen status, and that there was no obvious superiority between, FAI, their equation or direct measurement. Others, however, have found that direct measurement of FT by commercially available kit was more accurate in diagnosing raised FT in women with hirsutism than FAI or mathematical formula (Wilke et. al.1987).

Male sexuality can be divided into two discrete parts. The ability to sustain an erection, and ejaculate and sexual motivation. Kwan et.al.(1983) studied the effects of testosterone replacement on 6 hypogonadal men using a placebo controlled crossover method and found no difference between the men receiving testosterone replacement

and those who were not in erectile responses to erotic material presented in the laboratory. Untreated men, however, reported fewer sexual thoughts, acts, orgasms and spontaneous erections, and were noted to have reduced nocturnal penile tumescence. Kwan concluded that androgens were important for the motivational aspects of male sexual function. Bancroft et.al. (1983) studied 8 hypogonadal men and 8 age matched controls. Erections in response to erotic films were no different from those of the controls and were not changed by androgen replacement. Erections to self generated fantasy, however, were smaller and slower to develop in the hypogonadal men and did show significant improvement during androgen replacement. Bancroft makes the point that androgens were withdrawn for only short periods of time and more prolonged withdrawl might produce effects on erections in response to erotic material. In a latter study (O'Carroll et.al. 1985) 8 hypogonadal men were given oral testosterone undecanoate and a dose response relationship found between frequency of sexual thoughts and arousal accompanying those thoughts and sleep erections.

All of these studies have been performed on hypogonadal men. When young eugonadal men have been studied no correlation between androgen levels and sexual activity and thoughts have been found (Kraemer et.al.1976; Bancroft 1989; Gooren 1987; Sherwin 1988) this has led to the proposal that there is an androgen threshold above which increases in testosterone are behaviourally irrelevant. Salamimies et.al.(1982) studied 15 hypogonadal men and concluded that the level of total testosterone below which sexual function was impaired was between 6 nmol/l and 12 nmol/l, and noted that 4 men with TT in this range reported frequent erections and ejaculation which did not

increase with testosterone replacement. In a larger study Gooren (1987) examined the effects of testosterone replacement on a group of 26 hypogonadal men and 6 eugonadal medical students. He found that reduced androgen levels affected the subjective quality of sexual acts and the frequency of sexual thoughts, and observed that levels of total testosterone between 4 nmol/l and 6 nmol/l were neccessary for normal sexual interest in the hypogonadal men studied.

The extent to which androgens influence female sexual behavior remains unresolved. Udry et. al. (1986) showed that adolescent girls' first sexual experiences were strongly influenced by peer pressure and what was considered " suitable " by friends and family, hormonal status being secondary to these factors, whereas in boys rising testosterone levels consequent upon puberty correlated with sexual experimentation. One conclusion from this work is that female sexuality tends to be more rigorously controlled by society than male. Sherwin et. al. (1985) showed that testosterone supplementation in women after hysterectomy and oophorectomy, enhanced sexual desire, sexual fantasies, and arousal, but had no effect on coital frequency or satisfaction implying that testosterone was required to satisfy the cognitive aspects of sex rather than the purely physiological. Similar findings being reported in men (see above). Sherwin and her colleagues point out that the husbands of the women involved were not questioned, and suggested that once a sexual pattern has been established by couples that it might be difficult for one or other of the partners to change it against the wishes of the other. In addition some of the women involved may have been unused to the idea that they could initiate sexual activity. Another study of androgen levels and sexual activity in women

(Persky et. al. 1982) demonstrated that a group of post - menopausal women with significantly lower androgen levels than a group of pre - menopausal women had a lower frequency of intercourse than the younger women, despite there being no difference between the two groups in reported levels of sexual desire and arousal. This disparity between reported desire and frequency of intercourse may reflect an aging husbands diminished enthusiasm.

The pattern of changing hormone levels during the menstrual cycle and the advent of the oral contraceptive pill, which suppresses these has enabled workers to investigate the role of different hormones on female sexual interest. Adams et. al. (1978) noted a rise in female initiated sexual activity at the time of ovulation in women not using the OCP which was absent in those women taking the OCP. They concluded that the increase in sexual interest was probably due to the rise in oestrogens which occurs in the course of the menstrual cycle. Bancroft et. al. (1980) studied a group of 40 women all of whom took the OCP. 20 of these women had sexual problems which they ascribed to their use of the OCP. Plasma total testosterone, androstenedione, oestradiol and SHBG were measured. At the same time a behavioural assessment was made of each subject exploring aspects of physical arousal, their sexual attitudes towards themselves and their partner. Each subject also kept a diary of sexual activity and sexual thoughts. Although there were differences between the two groups in their behavioural assessment there were no differences in any of the hormonal perameters. Moreover when androstenedione was administered to the group complaining of dysfunction there was no difference in their sexual function dispite a rise in total testosterone. In a further study

(Bancroft et. al. 1991) plasma free testosterone levels were measured along with measures of sexual attitude and behavior. One of the instruments used in the behavioral part of the study being the SES scales. The investigators had predicted that FT would have an activating effect on sexual behavior. In reality this was only observed in those women taking the OCP for whom high FT levels were associated with more frequent sexual activity, and greater allowance of psychosexual stimuli as measured by SES 2. In addition those women using the OCP showed a more permissive sexual morality on SES 1, also to express less satisfaction with their relationship, and be more tolerant of the idea of extramarital involvement (SES 4). Bancroft and colleagues argue that the correlation between FT and some aspects of sexual behavior in OCP users and the absence of any correlations in women not taking the OCP may be explained by the fact that androgen effects on sexual behavior may be readily obscured by psychosocial influences. Bancroft also makes the point that these psychosocial factors should be controlled for in studies, and that these influences are likely to be different at different times in a every woman's life.

SEX HORMONE BINDING GLOBULIN (SHBG)

SHBG is a glycoprotein, synthesised in the liver, with a high affinity for testosterone, oestrogen, and oestradiol, in that order (Anderson 1974; Selby 1990). The metabolic clearance rate of those sex steroids which bind to SHBG is markedly lower than those which do not (Southren et. al. 1967), and it is thought that one of its actions in the protection of those sex steroids it binds from metabolism and excretion by the liver. Another of its actions is the modification of the rate of peripheral conversion of AND to testosterone, which is greater in individuals with low SHBG. Certain conditions are known to affect SHBG levels with high values found in thyrotoxicosis, hepatic cirrhosis in men, hypogonadism, and androgen insensitivity (Selby 1990). Low levels occur in association with myxoedema, hyperprolactinaemia, and androgen excess (Anderson 1974; Selby 1990).

In normal men approximately 44 % and in women 80 % of the available SHBG binding sites remain unoccupied. Over 99 % of available binding sites on albumin also remain unoccupied (Selby 1990), thus fluctuations in SHBG concentration can produce marked changes in both free and albumin bound hormone levels. It is thought that SHBG, and to a lesser extent albumin play a role in smoothing out these fluctuations, in addition to providing a reservoir of available hormone. Oestrogens are known to increase SHBG levels, and testosterone to lower them (Anderson 1974). Increased levels of oestrogen by increasing SHBG will cause a reduction in percentage unbound or free testosterone thus amplifying the oestrogen effect and causing a further rise in SHBG (Anderson

1974), the situation thought to pertain in normal women. In contrast in men testosterone diminishes SHBG levels increasing the percentage free testosterone, causing a further fall in SHBG. This servo mechanism proposed by Anderson (1974) is thought to be important in the maintenance of secondary sexual characteristics is both sexes.

SHBG levels are said not to vary during the menstrual cycle (Anderson 1974; Pearlman et al 1967; Kim et al 1976; Motohashi et al 1979; Odlind et al 1982), despite the well accepted observation that SHBG levels rise in pregnancy (Anderson 1974) and on administration of synthetic oestrogen (Pearlman et. al. 1967). Dowsett et. al. (1985), however found a significant rise in SHBG between follicular and luteal phases with the increment occuring between the day of ovulation and 2 days afterward. They found a significant correlation between preovulatory oestradiol levels and rise in SHBG. Of greater interest, however, is the observation made by Dowsett and colleagues of the effects this change in SHBG level would have on free testosterone. Using an equation not dissimilair to the one we used to compute free testosterone (TT:{% FT} = Log 8.52 -0.44 Log SHBG), they calculated that the effect of these changes would be a fall in free testosterone from 1.69% - 1.60%.

THE MENSTRUAL CYCLE

Menstruation punctuates the reproductive lives of women and is the product of a complex interplay between oestrogens, progesterone, LH and FSH. Day one of the menstrual cycle is taken as the first day on which bleeding occurs, and coincides with initiation of growth in one of the graafian follicles. During the early part of this follicular phase of the menstrual cycle ovarian output of oestradiol changes little, and LH and FSH are under negative feedback. Towards the end of this phase oestradiol, 17 hydroxy-progesterone, testosterone and AND rise. At mid - cycle there is a surge of oestradiol during which concentrations of the hormone increase by 200 - 400 %. Testosterone, AND and 17 hydroxy-progesterone also peak at this time. This precipitous rise in Oestradiol causes a surge of LH from the anterior pituitary by positive feedback, and ovulation takes place as a result (Johnson et. al. 1991). The second half of the menstrual cycle, known as the luteal phase is characterised by raised progesterone which peaks approximately 8 days after the LH surge. Oestradiol, and 17 hydroxy- progesterone levels also rise for a second time but do not reach the levels attained in the follicular phase. Testosterone and AND decline to their lowest levels, as do LH and FSH which are one again under negative feed back control, because of the reduction in oestradiol. If conception has not occurred by the end of the luteal phase both progesterone and oestradiol levels drop further, causing the endometrium to shed, and the woman to menstruate. The fall in oestradiol and progesterone allows LH and FSH to rise and thus initiate another cycle (Johnson et. al. 1991)

There are receptors for both oestrogens and progestagens in both the anterior pituitary and hypothalamus where there are large regional concentrations of these receptors in the pre - optic area, anterior hypothalamus and median eminence. Oestrogens and progestagens may exert their actions by changing the sensitivity of the gonadotrophs

to the GnRH pulses which arrive from the hypothalamus. Secondly they may alter the activity of the GnRH neurones in the hypothalamus (Johnson et. al. 1991). In men LH stimulates testosterone production in the Leydig cells, whilst FSH stimulates growth of the seminiferous tubules and the early stages of spermatogenesis. Because men do not exhibit a cyclical pattern of hormonal secretion like women they do not sustain an LH surge. Male gonadotrophin levels are controlled by testosterone which exerts a negative feedback effect on their secretion, with high levels of gonadotrophins being found in hypogonadism. Androgen receptors are present in both the anterior pituitary and hypothalamus. In rats the testosterone metabolite 5 α dihydrotestosterone has been shown to cause a significant fall in LH when implanted into the hypothalamus (Johnson et al 1991), a similar mechanism is thought to operate in man. Both LH and FSH are measured by radioimmunoassay.

AIMS OF STUDY

From the literature review it can be seen that many of the studies of sexual function in people with epilepsy were performed on individuals living in institutions or who had received surgical treatment for their epilepsy. Some of the studies in which testosterone levels were ascertained were undertaken before the more recent findings that men can have normal libido in the face of a wide range of testosterone levels and that psychosocial factors may play as much a role in female sexuality as androgen levels. Many of the studies used ad hoc questionnaires which had not been validated, others were undertaken in cultures different from our own. Others concentrated purely on actual sexual activity without trying to quantify the effect if any of the particular social pressures faced by people with epilepsy. The aim of this study was to get as all round a view of our subjects sexuality, in relation to their epilepsy, as possible, and were defined as follows.

1. To examine attitudes to sexual morality, and premarital sexual experience of people with epilepsy compared with a randomly selected control group.
2. To ascertain whether people with epilepsy differ in their openness to psychosexual stimulation, in particular their arousability by erotic imagery and their feelings upon being exposed to such imagery.
3. To compare the group with epilepy's enjoyment of sexual interaction and preferred frequency of intercourse compared with the control group.
4. To examine the effects of antiepileptic drugs on sexual arousability

5. To determine the effects, if any, of seizure type, frequency and age at onset of epilepsy on indices of arousability, and preferred frequency of intercourse.
6. To examine the relationship, if any, between free testosterone and preferred frequency of intercourse and enjoyment of sexual interaction.
7. To examine the effects in men of individual drugs on sex hormone binding globulin, total and free testosterone levels, and in women to study the effects of antiepileptic drugs on sex hormone binding globulin, and total and free testosterone levels.

METHODS

Description of Epilepsy Clinic at Western Infirmary Glasgow

The patients who kindly consented to help in this study attended the Epilepsy Clinic at the Western Infirmary Glasgow. This is an integral part of the Epilepsy Research Unit, and is sited in the University Department of Medicine and Therapeutics. It is staffed by one consultant clinical pharmacologist, a senior registrar, a senior house officer and two clinical research fellows. At the time during which this study was performed one thousand patients had been referred 52% of which were female. 52% were 30 years of age or younger at time of referral. 51% were married, 40% single, 6% were divorced, and 3% were widows or widowers. 760 of the patients lived in the Glasgow area, the remaining 240 (24%) had been referred from outwith the Greater Glasgow Health Board area.

The majority of patients (53%) had been referred by their general practioners. Most of the rest (45%) came from other hospital practioners. Of these, 51% were sent to the clinic by a general physician, of which 36% were direct referrals from the Accident and Emergency department at the Western Infirmary. Smaller numbers of tertiary referrals came from neurologists (6%), neurosurgeons (2%), obstetricians (2%) psychiatrists (2%), paediatricians (2%) and from epilepsy related institutions. In most cases where an eye witness account was available it was possible to classify the siezure type. Generalised tonic-clonic seizures occured in 52% of the patients. Just over half (52%) of these were thought to be primary generalised the rest (48%) were recognised as being secondary generalised. Simple and complex partial seizures occurred in 13% and 34%

respectively. More uncommon seizure types such as atonic, myoclonic, and absence seizures occurred in 3%, 3% and 1% of patients respectively. In addition 88 (8.8%) were thought to have pseudoseizures.(M.J. Brodie 1992)

PATIENTS

159 women all of whom lived independently in the community were recruited. All were judged to be compliant with their AED regime by dint of drug levels obtained on the day of the study and from previous clinic visits, and no change to their AED therapy had been made in the previous three months. This cohort of subjects comprised the "treated group". A further 36 women who had either been recently diagnosed as having epilepsy, and had not been commenced on AED therapy or who were undergoing withdrawal of drugs due to remission were also recruited, and comprised the "untreated" group.

Of these 195 women 110 suffered from complex partial seizures (CPS) with or without secondary generalisation, 56 had primary generalised epilepsy (PGE) and 28 had seizures which were undefined (including simple partial seizures, focal motor seizures).

48 women were recruited consecutively from relatives accompanying patients to the clinic, patients in the adjacent orthopaedic clinic, and hospital staff not known to the investigators (see appendix 1 for description of female control group)

Of these 243 women 65 took the oral contraceptive pill (OCP) and were thus excluded from hormonal analysis, and a further 47 were unable to recall the first day of their last period or had irregular periods they to were excluded.

153 men were recruited. Of these 18 received VPA monotherapy, 31 CBZ monotherapy, 21 PHT monotherapy, and 48 polytherapy with more than one of the aforementioned AEDs. For the purposes of the demographic data and sexuality experience scale analysis these men comprised the "treated" group. 34 men who were either newly diagnosed, or undergoing a trial withdrawal of AEDs were also recruited, this was the "untreated" group. A control group of 45 men was also recruited in the same manner as that of the women (see appendix 1 for description of male control group). 75 of the men suffered from CPS with or without secondary generalisation, 86 PGE and 26 were assigned to the undefined group as for the women.

All of the men lived independantly in the community and were judged to be compliant with their AEDs in the same way as for the women. None of the patients or controls suffered from a progressive neurological disease and all were biochemically euthyroid.

DEMOGRAPHIC DATA

The following data were obtained by inspection of case notes and by asking each subject to fill in a short questionnaire. Each subject was asked about marital status, with the respondant being considered "married" if they had been cohabiting for one or more years. Whether or not they had received further education. Religious affiliation with each person asked to rate the strength of that affiliation as 1 if they attended church regularly- at least twice a month- and considered their religious beliefs an important part of their life. If they attended less frequently and felt religion was of significance only at certain times e.g Christmas or at times of illness etc then they were asked to record their affiliation as 2.

From the case records the individuals seizure type, age at onset of epilepsy, drug therapy, and number of seizures in preceding three months. Women were asked if they were pregnant or taking the OCP.

SEXUALITY EXPERIENCE SCALES (SES)

The Sexuality Experience Scales devised by Frenken and Vennix (1981) were chosen for a number of reasons. Firstly it had been validated in a number of field trials, and had been shown to be useful in assessing sexual dysfunction. Secondly it acknowledged that sexuality is multidimensional and subject to the individuals background, beliefs and the society in which he/she lives. Thirdly it was easy to use, and the concepts underlying the scales easy to grasp for a clinician not trained in psychology or sexual research. Lastly it was simple to administer by means of a personal computer which automatically calculated each subjects score, and stored it. The investigator felt these advantages would outweigh the potential disadvantage of a written questionnaire favouring those with a high standard of literacy as opposed to those who did not.

Before each subject was shown how to use the computer the questionnaire they had filled out giving details or marital status, religious affiliation etc was inspected by one of the research assistants or myself to ensure all questions had been answered . This was also a way of checking that the respondents were literate enough to cope with the computerised questions. None of the respondents failed this albeit crude test of literacy.

The fundamental concept behind the construction of the four Sexuality Experience Scales is that of " rejection verses acceptance of sexuality ", and the assumption that everyone occupies some point along this hypothetical scale. In formulating the scales Frenken and Vennix concentrated on three broad aspects of sexual behaviour.

1) Behaviour and behavioural tendencies in terms of approach and avoidance

2) Experiences in terms of negative and positive feelings

3) Opinions and values in terms of positive or negative judgements of various aspects of sexuality.

Initially 182 questions framed around the following themes

Experiencing ones owns body

Experience of partners body

Sexual stimulation

Communication with others about sex

The relative importance of sexuality

Sexual relationships before and outside marriage

Behaviour and experiences in situations of sexual play

Behaviour and experiences associated with coitus, orgasmic adequacy and experiences related to orgasm

In the preliminary study 52 men and 55 women all of whom were under the age of 55 and married from what Frenken and Vennix described as a " broad " middle class were interviewed using these questions. Principal component analysis with oblique analysis rotation over the items produced three factors which consistently emerged. These can be seen as three dimensions with opposite poles. They are -:

Permissiveness versus restrictivness in sexual morality.
Seeking and allowing versus the avoidance of psychosexual stimulation.
A tendency to seek or avoid sexual interaction with one's partner.

A second study involving 250 married men and women all of whom were under the age of 55 and from a " broad " middle class was performed in which 76 questions pertaining to these three dimensions were used. In addition a further 24 questions designed to assess the individuals attraction to marriage were tested on this group. Principal component analysis showed that the results from the first study were replicated. The questions were then divided into the four SES scales which are described in greater detail below.

A further 250 men and women under the age of 55 were presented with the questionnaire, a high degree of correlation in scores was found between these two populations, and from these two studies a set of normative data was drawn up (See Appendix 4).

Further studies were performed on groups of individuals seeking help for psychosexual dysfunction. These individuals attained significantly different scores from the randomly selected populations. From this normative data for men and women complaining of psychosexual dysfunction was compiled.

Factor Analysis

The principals of Factor analysis and Principal Component analysis by which Frenken and Vennix arrived at their questionnaire will be briefly explained. Concepts such as patience and love cannot, like temperature and blood pressure, be measured on a scale. Rather they are constructs that unify characteristic responses to a related group of variables. Answers of strongly agree to questions like he/she spends all his/her time with me, sends me flowers every day, can't keep his/her hands off me would lead to the conclusion that the love factor is present. Whereas answers of strongly agree to questions like he/she avoids me, can't recall my name, never phone's leads to the very opposite conclusion. Love is not a single measurable entity but a construct derived from measuring other directly observable variables. Identification of underlying factors or dimensions simplifies the description of complex concepts like love, creativity etc.
Factor analysis is the statistical technique used to identify a small number of factors that can be used to represent relationships among sets of many interrelated variables. For example the factors which make the individual attracted to marriage.

The first step in factor analysis is the construction of a correlation matrix for all the variables measured. From this variables with a high degree of correlation can be identified. As the goal of the analysis is to find factors that explain these correlation's these variables must be related to one another for the factor model to be appropriate. Thus variables with low or no correlation are presumed not to have the ' factor' in common.

In the second step, principal components analysis, linear combinations of observed variables are formed. The first principal component is the combination that accounts for the largest variance in the sample. The second principal component accounts for the

next largest amount of variance and is uncorrelated to the first. Ideally the fewer principal components the better.

Thus by the end of the factor extraction phase the number of common factors required to describe the data adequately is determined.

Although the factor matrix indicates the relationship between factors and variables it is often difficult to identify meaningful factors based on the matrix with most factors appearing to correlate with many variables. Rotation is designed to make the initial matrix easier to understand. Rotation is aimed at reducing the number of variables with moderate correlation's with several factors. After rotation variables highly correlated with single factors are more visible on the factor matrix.

Put simply after rotation one would expect to see a high degree of correlation between the variables can' t keep his/ her hands off me , spends all his/her time with me, sends me flowers. This would be described as the love factor.

SEXUALITY EXPERIENCE SCALE 1 (SES 1) Sexual Morality Scale

The questions on this scale refer to attitudes, and values pertaining to sexual behaviour in marriage, pre-marital sex, and what is permissible for the young to know and practise about sex. A high score reflects a restrictive sexual morality or avoidance. A low score indicates a more permissive morality and implies acceptance and approval of sexuality. Frenken and Vennix found women tended to attain higher scores than men, as did those with a strong religious affiliation. On the other hand those men and women who had gone on to higher education tended to have lower scores.

Within SES 1 there are three subscales aimed at examing different aspects of sexual morality.

MS	Sexual morality within marriage	Permissive -	Restrictive +
PS	Premarital sexual experience	High -	Low +
PA	Premarital sexual attitude (men)	Permissive -	Restrictive +
SS	Sexual socialisation (women)	Permissive -	Restrictive +

SEXUALITY EXPERIENCE SCALE 2 (SES 2) Psychosexual Stimulation

The items on this scale refer to the extent that a person will seek or allow as opposed to avoiding or rejecting psychosexual stimulation whether external or imaginary. The different situations asked about in this scale are rated by the respondent from " very pleasant " to " very unpleasant ". A high score implies low psychosexual arousability, or avoidance. A low score means high allowance of psychosexual stimulation.

Once again women have been found to attain higher scores than men. For both sexes those with a strong religious affiliation tend to achieve higher scores than those without religious beliefs. Both men and women who went on to higher education are more likely to have lower scores than those who had not.

There are three subscales within SES 2

AE	Arousal by erotic imagery	Seeking -	Avoiding +
EE	Evaluation of exposure to erotic imagery	Like -	Dislike +
IS	Interpersonal sexual attraction	High -	Low +

Both SES-1 and SES-2 were administered to all the subjects.

The remaining scales, SES-3 and 4 and their subscales were answered by those in a stable heterosexual relationship.

SEXUALITY EXPERIENCE SCALE 3 (SES 3) The Sexual Motivation Scale

The items on this scale refer to the sexual interaction with the respondents long-term partner. It explores the intensity of arousal during sex, the frequency, both preferred and actual of intercourse, and the occurrence of ejaculatory and orgasmic difficulties. The scale asks questions about certain aspects of sex play and asks the respondent to rate them on a scale of " pleasant " to " not at all pleasant ". Lastly it examines the occurrence and frequency of emotions during intercourse. Inquiring of the subject if they have feelings of aversion, fear, apathy, tension or disappointment towards the sex act. A high score means a strong avoidance tendency or " sexual aversion ". A low score denotes a strong approach tendency or sexual appetite.

Women tend to attain higher scores than men as do older men and women who have been in long term relationships.

There are five subscales within SES 3 which are delineated below. The first two were of particular importance to the author because of interested in establishing a relationship, if any between free testosterone levels and sexual activity.

EP	Enjoyment potential of sexual interaction	Strong - Weak +
FI	Preferred frequency of intercourse	High - Low +
OE	Orgasm adequacy during intercourse and its evaluation	Strong - Weak +
LF	Length of Foreplay	Long - Short +
SI	Sexual Inhibition (Women only)	Strong - Weak +
II	Inhibition During intercourse	Absent - Strong +

SEXUALITY EXPERIENCE SCALE 4 (SES-4) The Attraction to Marriage Scale

The items on this scale pertain to the reasons why an individual would want to continue in a marriage. They are threefold: Social and emotional attraction to the partner, the social and moral unacceptability of ending the relationship, and the sexual attraction to the partner. A high score implies strong social, emotional and sexual bonds with the partner and social as well as moral resistance to ending the marriage.

Women and individuals with a strong religious affiliation tend to achieve higher scores.

There are three subscales within SES 4

MS	Marital Satisfaction	High -	Low +
AE	Attitude to Extramarital Involvement	Restrictive -	Permissive +
ES	Evaluation of Partner as Sexual Partner	Positive -	Negative +

It is possible to calculate a weighted score for the scales which is more accurate than the raw data. This is essential for calculation of the subscale scores because they contain few items.

In this study the raw scores for SES 1,2 3 and 4 are used to give an overview of each scale and the weighted subscale scores employed to examine certain aspects of the subjects sexual response. Following the method of Frenken and Vennix (1981) the subcale scores are normalised, giving a mean score of zero for their normative sample.

The questionnaire was administered to all subjects by means of a personal computer equipped with a joystick attachment. This method was chosen because the computer calculated the unweighted scores thus saving time, and more importantly it was impossible for the respondant to skip questions as each new question came on screen only on completion of the previous one. Each person not familiar with computers was given instruction in its use and allowed to answer one or two general knowledge questions which had been added to the questionnaire for this purpose. The subject was then left alone in the room where the computer was situated. As each question was answered the next automatically appeared on screen. The whole questionnaire took on average 15 -20 minutes to complete depending on the subject. Each person after the initial period of instruction and practise was told by the investigator that should they

encounter any problems with the computer or find a question unclear they should not feel embarrassed by summoning the investigator to explain. There were surprisingly few people who required assistance of any kind. The SES scales were designed for and validated on heterosexuals. To get round this problem each subject was told that the questions on the computer were designed for people married to or living with a man / women depending on the sex of the subject being addressed. Those who were not in a relationship at the time of the study were told that the questions they were being asked to respond to were for heterosexuals .

HORMONE ASSAYS

Total testosterone, androstenedione, and dehydroepiandosterone sulphate were all determined by standard radioimmunoassay (Ismail et.al.1986: Connell et.al. 1984) all had a mean interassay coefficient of variation of 5-10%.

SHBG was measured by the DELFIA ^{TM} . Two - site immunoflurometric assay (Selby 1990).

Free testosterone was calculated after the method of Nanjee and Wheeler (1985)

Free testosterone (pM) = Total Testosterone times % Free Testosterone times 1000 divided by 100.

Where % Free Testosterone = -2.38 log 10 { SHBG (nM) } + 6.11.

This equation correlated very closely with measured free testosterone ($r = 0.84$ $P< 0.0001$) in the study.

This equation has the advantage that it uses two easily measured parameters i e TT and SHBG, but it does depend on both of these substances being accurately measured. It does not take into account testosterone bound to albumin or cortisol binding protein (Nanjee et al 1985) . This method has been found to give free testosterone results which correlate closely with directly measured using the antibody - coated tube radioimmunoassay method (Wilke et.al 1987;Cheng et.al. 1986: Dechaud et.al.1989).

All hormone assays were performed in the endorcrine laboratory of Glasgow Royal Infirmary under the supervision of Dr G. Beastall.

STATISTICAL METHODS

All demographic, hormonal, drug level and SES scores were transferred into a standard personal computer. All calculations were performed by Minitab (release 10) a standard statistics package designed for personal computers. Parametric statistical tests in the form of one analysis of variance (ANOVA) and twosample t test were applied where the data exhibited a Gaussian distribution. P value of ≤ 0.05 was taken as significant in all calculations. Multiple comparisons using Fishers equation were automatically calculated by Minitab.

When data was not normally distributed it was transformed into logarithmic values and if the subsequent values were in a Gaussian distribution ANOVA was employed. If this failed then the appropriate non-parametric test was employed. Simple linear regression and Pearson correlation coefficient were calculated by Minitab to ascertain the relationship if any between AE scores and AED levels and number of seizures in the preceding three months. Similarly for FI scores and seizure frequency in preceding three months.

Multiple regression to examine the effects if any on SES responses by independant variables was performed on Minitab using dummy variables where categoriacal data was involved (see statistical appendix). Some examples of plots of residuals are given to demonstrate a normal distribution.

Categorical data was analysed by Minitab using the chisquare test. Statistical calculations as performed on Minitab, along with frequency histograms, details of logarithmic transformation and dummy variables used in multiple regression are given in the statistics appendix at the end of this thesis. The calculations involved in simple regression and chisquare tests are given in the main text either immediatly below the scatterplots in the case of regression or as part of the frequency table in the case of chisquare tests.

RESULTS

RESULTS OF DEMOGRAPHIC DATA AND SES 1

The male treated group was significantly older than the control and untreated groups (Table 3 82), whilst the female untreated group was significantly older at time of study than both the control and treated group (Table 4 82). Those men in the CPS group were significantly older than the controls (Table 5 82) , but there was no significant difference in age between any of the seizure type groups and controls in the female sample (Table 6 83). There was no significant difference in age at onset of epilepsy between the untreated and treated male groups (Table 7 83), but the age at onset of the female untreated group was significantly older than that of the treated group (Table 8 83). Both female and male control groups were significantly more likely to have had further education than the untreated and treated groups (Table 9,Table 10 84). There were no significant differences between the control, untreated, and treated groups for marital status in either sex (Table 11 84,Table 12 85), nor were there any significant differences in marital status between the different seizure type groups for either men or women (Table 13 85, Table 14, 86).

Table 3: AGE AT TIME OF STUDY FOR MEN

GROUP	N	MEAN (Years)	STANDARD DEVIATION
CONTROL	45	27.8	7.1
UNTREATED	34	27.6	8.1
TREATED	153	32.1*	9.1

*** P ≤ 0.05 Compared with Control and Untreated Groups (ANOVA)**

Table 4: AGE AT TIME OF STUDY FOR WOMEN

GROUP	N	MEAN (Years)	STD
CONTROL	48	30.2	9.3
UNTREATED	36	25.3*	6.24
TREATED	159	29.2	8.4

P = 0.018

*** P ≤ 0.05 Compared with Control and Treated Groups (ANOVA)**

Table 5: AGE of MALE SEIZURE TYPE GROUPS

GROUP	N	MEAN (Years)	STANDARD DEVIATION
Complex Partial Seizures	75	33.9 *	9.1
Primary Generalised Epilepsy	86	30.4	9.7
Undefined	26	30.4	7.0
Control	45	27.8	7.1

*** P ≤ 0.05 Compared with Control Group (ANOVA)**

Table 6: AGE OF FEMALE SEIZURE TYPE GROUPS

GROUP	N	MEAN (Years)	STD(yrs)
Complex Partial Seizures	110	29.3	8.3
Primary Generalised Epilepsy	56	27.5	7.7
Undefined	28	28.4	8.4
Control	48	29.6	9.2

ANOVA not significant

Table 7: AGE AT ONSET OF EPILEPSY FOR MEN

GROUP	N	MEAN (Years)	STANDARD DEVIATION
UNTREATED	34	21.9	10.17
TREATED	153	20.1	12.44

P = 0.467 (ANOVA)

Table 8: AGE AT ONSET OF EPILEPSY FOR WOMEN

GROUP	N	MEAN (Years)	STD
UNTREATED	36	18.4	7.40
TREATED	159	16.7	9.50

$P \leq 0.05$(ANOVA)

Table 9: FURTHER EDUCATION IN MALE GROUP

GROUPS	FURTHER EDUCATION	NO FURTHER EDUCATION	Total
Control (Expected)	28 (20.95)	17 (24.05)	45
Untreated (Expected)	15 (15.83)	19 (18.17)	34
Treated (Expected)	64 (71.22)	89 (81.78)	153

CHI-SQUARE = 7.242 DF = 2 P = 0.025

Table 10:FURTHER EDUCATION IN FEMALE GROUP

GROUPS	FURTHER EDUCATION	NO FURTHER EDUCATION	Total
Control (Expected)	32 (20.1)	16 (27.8)	48
Untreated (Expected)	16 (15.0)	20 (20.9)	36
Treated (Expected)	54 (65.8)	105 (91.2)	159

CHI-SQUARE = 19.28 DF = 2 P = 0.005

Table 11: MALE MARITAL STATUS

GROUPS	MARRIED	NOT MARRIED	Total
Control (Expected)	17 (19.78)	28 (25.22)	45
Untreated (Expected)	12 (14.95)	22 (19.05)	34
Treated (Expected)	73 (67.27)	80 (85.73)	153

CHI-SQUARE = 2.609 DF = 2 P = 0.1

Table 12: FEMALE MARITAL STATUS

GROUPS	MARRIED	NOT MARRIED	Total
Control (Expected)	20 (18.57)	28 (29.4)	48
Untreated (Expected)	10 (13.9)	26 (22.0)	36
Treated (Expected)	65 (61.5)	94 (97.4)	159

CHI-SQUARE = 2.150 DF = 2 P =0.1

Table 13: MARITAL STATUS IN DIFFERENT MALE SEIZURE GROUPS

GROUPS	MARRIED	NOT MARRIED	Total
Complex Partial Seizures (Expected)	38 (36.36)	37 (38.64)	75
Primary Generalised Epilepsy (Expected)	36 (41.00)	50 (45.00)	86
Undefined (Expected)	11 (12.64)	15 (13.76)	26

CHI-SQUARE = 0.767 DF = 2 P = 0.90

Table 14: MARITAL STATUS IN DIFFERENT FEMALE SEIZURE GROUPS

GROUPS	MARRIED	NOT MARRIED	Total
Complex Partial Seizures	43	67	110
(Expected)	(42.3)	(65.6)	
Primary Generalised Epilepsy	16	40	56
(Expected)	(22.3)	(34.6)	
Undefined	16	12	28
(Expected)	(13.3)	(20.6)	

CHI-SQUARE = 0.767 DF = 2 P = 0.1

Age at onset of epilepsy appeared to influence the marital status of the male group with men who developed seizures significantly less likely to be married than those who developed disease later in life (Table 15 86). Age at onset did not appear to affect the marital status of women (Table 16 87) although those women sustaining their first seizure in the first two decades had lower rates of marriage than women developing seizures later on.

Table 15: RATES OF MARRIAGE FOR MALE AGE AT ONSET GROUPS

Age at Onset	Unmarried	Married	Percentage Married (of each age at onset group)
0-10 years	26	13	
(Expected)	(20.15)	(16.86)	33%
11-20 years	44	17	
(Expected)	(32.67)	(27.33)	27.8%
21-30 years	21	31	
(Expected)	(27.22)	(22.78)	59.0%
31 years and over	11	25	
(Expected)	(17.97)	(15.03)	71%
Total	102	86	

CHI-SQUARE = 25.23 DF = 3 P = 0.00

Table 16: RATES OF MARRIAGE FOR FEMALE AGE AT ONSET GROUPS

Age at Onset	Unmarried	Married	Percentage Married (of each age at onset group)
0-10 years (Expected)	26	12	31.5%
11-20 years (Expected)	64	34	34.6%
21-30 years (Expected)	21	19	47.5%
31 years and over (Expected)	9	10	50%
Total	120	75	

CHI-SQUARE = 1.699 DF = 3 P = 0.1

With regard to SES 1 (Sexual Morality Scale) scores the male treated group had a significantly higher SES 1 (unweighted score) than the control group (Table 17 88), suggesting a restrictive morality. The men in the treated group had a higher mean MS (sexual morality within marriage) subscale score than the controls, but exhibited no significant differences in the other two subscales, although there was a trend for the treated group to attain higher scores.

Table 17: MALE SES 1(UNWEIGHTED) AND SES 1 SUBSCALES (WEIGHTED)

GROUP	N	SES (Unweighted) Mean (STD)	MS Mean (STD)	PS Mean (STD)	PA Mean STD)
CONTROL	45	49.9 (11.65)	-0.17 (0.40)	-0.46 (0.99)	-0.03 (0.34)
UNTREATED	34	54.5 (12.96)	0.02 (0.45)	-0.46 (0.81)	-0.01 (0.44)
TREATED	153	57.6 (14.32)*	0.16 (0.53) *	-0.43 (0.92)	-0.06 (0.40)

*** P ≤ 0.05 Compared with Control Group (ANOVA)**

MS Sexual Morality Within Marriage
PS Premarital Sexual Experience and Attitude
PA Premarital Sexual Attitude

The female treated group had a significantly higher mean SES 1 unweighted score than both the control and untreated groups. The same pattern was seen in both MS and SS (sexual socialisation morality) subscale scores. The treated group had a significantly higher PS score than the untreated group (Table 18 89).

Table 18:FEMALE SES 1(UNWEIGHTED) AND SES 1 SUBSCALES (WEIGHTED)

GROUP	N	SES 1 Mean (STD)	MS Mean (STD)	PS Mean (STD)	SS Mean STD)
CONTROL	48	54.6 (14.4)	-0.16 (0.55)	-0.32 (0.72)	-0.31 (0.52)
UNTREATED	36	54.3 (16.3)	-0.11 (0.11)	-0.40 (0.77)	-0.32 (0.58)
TREATED	159	63.3 (14.9)*	0.16 (0.55)*	-0.09 (0.73)•	-0.03 (0.63)♣

*** P ≤ 0.05 Compared with Control and Untreated Groups(ANOVA)**
• P ≤ 0.05 Compared Untreated Group(ANOVA)
♣ P ≤ 0.05 Compared with Control and Untreated Groups(ANOVA)

MS Sexual Morality Within Marriage
PS Premarital Sexual Experience and Attitude
SS Sexual Socialisation Morality

Multiple regression on four variables (age at time of study, further education, religious affiliation and marital status) was performed for SES 1 scores. For the men further education had a significant effect on unweighted SES 1 scores as well as on the MS (sexual morality within marriage) and PA (premarital sexual attitude) subscale scores (Table 19 91). None of these four variables were predictive for female SES 1 unweighted scores. Further education exerted a significant effect on both MS and SS (sexual socialisation) subscales, and religious affiliation on the PS (premarital sexual experience) subscale, age at time of study just missed significance on the MS subscale (Table 20 92)

Table 19:EFFECTS OF INDEPENDENT VARIABLES ON MALE SES 1 (UNWEIGHTED) AND SES 1 SUBSCALES (WEIGHTED)

INDEPENDENT VARIABLES				
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**	**MARITAL STATUS**
SES 1	β coefficient = 0.21 P= 0.056	β coefficient = -10.8 P= 0.00	β coefficient = 2.99 P= 0.086	β coefficient = -1.57 P= 0.434
PS	β coefficient = -0.003 P= 0.739	β coefficient =-0.034 P= 0.803	β coefficient = 0.053 P= 0.700	β coefficient = -0.199 P= 0.210
MS	β coefficient = 0.008 P= 0.080	β coefficient = -0.54 P= 0.05	β coefficient = 0.019 P= 0.792	β coefficient = -0.088 P= 0.305
PA	β coefficient = 0.005 P= 0.188	β coefficient =- 0.327 P= 0.05	β coefficient = -0.030 P= 0.626	β coefficient = -0.029 P= 0.680

MS Sexual Morality Within Marriage
PS Premarital Sexual Experience and Attitude
PA Premarital Sexual Attitude

See Statistical Appendix for explanation of dummy variables

Table 20:EFFECTS OF INDEPENDENT VARIABLES ON FEMALE SES 1 (UNWEIGHTED) AND SES 1 SUBSCALES (WEIGHTED)

INDEPENDENT VARIABLES				
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**	**MARITAL STATUS**
SES 1	β coefficient = 0.204 P= 0.12	β coefficient = -3.54 P= 0.086	β coefficient = 3.00 P= 0.16	β coefficient = -1.21 P= 0.59
MS	β coefficient =0.01 P=0.055	β coefficient =-0.25 P= 0.05	β coefficient =0.40 P=0.79	β coefficient =-0.04 P=0.570
PS	β coefficient =0.00 P=0.143	β coefficient =0.02 P=0.805	β coefficient =0.65 P=0.05	β coefficient =0.01 P=0.923
SS	β coefficient =0.00 P=0.848	β coefficient =-0.18 P=0.05	β coefficient =-0.02 P=0.801	β coefficient =-0.03 P=0.714

MS = Sexual Morality Within Marriage
PS = Premarital Sexual Experience
SS = Sexual Socialisation

See Statistical Appendix for explanation of dummy variables

RESULTS OF SES 2 PSYCHOSEXUAL STIMULATION SCALE

Both the treated and untreated male groups had significantly higher SES 2 unweighted scores, than the controls implying that they were less open to psychosexual stimulation. Examination of the subscales showed that the untreated group had a significantly higher IS (interpersonal sexual attraction) mean score compared with the controls and that both the untreated and treated groups had significantly higher mean AE (arousal by erotic imagery) scores than the controls. There were no significant differences on the EE (evaluation of exposure to erotic imagery) subscale(Table 21 94)

Table 21: MALE SES 2 (UNWEIGHTED) AND SES 2 SUBSCALES (WEIGHTED)

GROUP	N	SES 2 Mean (STD)	IS Mean (STD)	EE Mean (STD)	AE Mean (STD)
CONTROL	45	35.4 (6.88)	-0.83 (0.55)	-0.38 (0.62)	-0.92 (0.96)
UNTREATED	34	41.5 (11.1) *	-0.38 (0.80) *	-0.02 (0.75)	-0.41 (1.06) *
TREATED	153	40.2 (9.0) *	-0.62 (0.70)	-0.30 (0.80)	-0.45 (1.09) *

*** P ≤ 0.05 Compared With Control (ANOVA)**

ANOVA EE P = 0.114
IS Interpersonal Sexual Attraction
EE Evaluation of Exposure to Erotic Imagery
AE Arousal By Erotic Imagery

Multiple regression of 4 variables was performed. For the SES 2 unweighted scores further education showed a significant interaction. Further education also had a significant predictive effect on the AE subscale scores. Age at time of study, religious affiliation and marital status appeared to have no predictive value on SES 2 and subscales (Table 22 95)

Table 22 : EFFECTS OF INDEPENDENT VARIABLES ON MALE SES 2 (UNWEIGHTED) AND SES 2 SUBSCALES

INDEPENDENT VARIABLES				
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**	**MARITAL STATUS**
SES 2	β coefficient = 0.019 P= 0.817	β coefficient = -4.40 P= 0.05	β coefficient = 0.973 P= 0.438	β coefficient = 0.648 P= 0.655
AE	β coefficient = -0.010 P= 0.313	β coefficient = -0.54 P= 0.05	β coefficient = -0.024 P= 0.875	β coefficient = 0.136 P= 0.441
EE	β coefficient = -0.002 P= 0.777	β coefficient = 0.015 P= 0.893	β coefficient = 0.125 P= 0.300	β coefficient = -0.069 P= 0.605
IS	β coefficient = 0.004 P= 0.522	β coefficient = -0.134 P= 0.187	β coefficient = 0.123 P= 0.277	β coefficient = 0.172 P= 0.142

See Statistical Appendix for explanation of dummy variables

The women in the treated group had a significantly higher mean SES 2 (unweighted) score than the control group. The treated group had significantly higher mean IS and EE scores than the controls. There were no significant differences on the AE subscale. Thus it appeared women receiving AEDs rated exposure to erotic imagery less positively than the controls and reported lower levels of interpersonal sexual attraction (Table 23 96).

Table 23:FEMALE SES 2 (UNWEIGHTED) AND SES 2 SUBSCALES (WEIGHTED)

GROUP	N	IS Mean (STD)	EE Mean (STD)	AE Mean (STD)
CONTROL	48	-0.64 (0.58)	-0.18 (0.76)	-0.36 (0.96)
UNTREATED	36	-0.49 (0.81)	-0.10 (0.81)	-0.32 (1.2)
TREATED	159	-0.23 (0.79)*	-0.23 (0.79) *	0.07 (1.05)

*** P ≤ 0.05 Compared With Control (ANOVA)**

ANOVA AE Not Significant

IS Interpersonal Sexual Attraction
EE Evaluation of Exposure to Erotic Imagery
AE Arousal By Erotic Imagery

Multiple regression of the same four independent variables as were examined for the men showed that none of these variables had a predictive effect for female SES 2 unweighted scores. Further education and religious affiliation, however, had a predictive effect on the IS subscale score (Table 24 97).

Table 24: EFFECTS OF INDEPENDENT VARIABLES ON FEMALE SES 2 (UNWEIGHTED) AND SES 2 SUBSCALES

INDEPENDENT VARIABLES				
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**	**MARITAL STATUS**
SES 2	β coefficient = 0.086 P= 0.255	β coefficient = -1.207 P= 0.308	β coefficient = -0.142 P= 0.908	β coefficient = -0.163 P= 0.90
AE	β coefficient = 0.00 P= 0.53	β coefficient = -0.12 P= 0.384	β coefficient = -0.13 P= 0.358	β coefficient = -0.12 P= 0.432
IS	β coefficient = 0.01 P= 0.117	β coefficient = -0.23 P= 0.05	β coefficient = 0.48 P= 0.05	β coefficient = 0.06 P= 0.546
EE	β coefficient = 0.00 P= 0.337	β coefficient = -0.02 P= 0.822	β coefficient = -0.05 P= 0.621	β coefficient = -0.03 P= 0.763

RESULTS OF SES 3 SEXUAL MOTIVATION SCALE

SES 3 was administered only to those men and women in a stable heterosexual relationship. There were no significant differences between the male study groups on SES 3 unweighted scores, neither were there any significant differences between the three study groups on any of the subscale scores in particular the EP (enjoyment potential of sexual intercourse) and FI (preferred frequency of intercourse) subscales (Table 25 100). There were no noticeable trends between the groups. The female treated group had a significantly higher mean SES 3 score than both the controls and untreated groups implying than they were less allowing of psychosexual stimulation. There were no significant differences between the three female study groups on the SES 3 subscales with the exception of the OE where the treated group had a significantly higher mean score than the control group suggesting that they rated orgasm during intercourse as less pleasurable than the controls (Table 26 101).

Multiple regression using three independent variables, age at time of study, further education and religious affiliation was performed. Age had a significant predictive effect on the male SES 3 unweighted scores, however, these three variables had no significant predictive effects on any of the subscale scores (Table 27 102). For the women further education had a significant predictive effect on SES 3 unweighted scores, but none of the three independent variables significantly influenced the SES 3 subscale scores (Table 28 103)

Table 25: **MALE SES 3 (UNWEIGHTED) AND SUBSCALE SCORES**

GROUP	N	SES 3 Mean (STD)	FI Mean (STD)	EP Mean (STD)	II Mean (STD)	LF Mean (STD)	OE Mean (STD)
CONTROL	17	46.5 (12.38)	0.19 (0.74)	0.97 (0.20)	1.31 (0.80)	-0.25 (1.23)	1.68 (0.73)
UNTREATED	12	45.8 (9.18)	0.56 (0.94)	0.86 (0.21)	0.91 (0.48)	-0.72 (1.43)	1.61 (0.73)
TREATED	73	53.3 (17.16)	0.24 (0.85)	0.89 (0.40)	1.53 (0.94)	-0.27 (1.52)	1.80 (0.73)

ANOVA of Subscales No Significant Differences

FI Preferred Frequency of Sexual Intercourse
EP Enjoyment Potential of Sexual Interaction.
II Inhibition During Intercourse
LF Length of Foreplay
OE Orgasm Adequacy During Intercourse and Its Evaluation

Table 26: FEMALE SES 3 (UNWEIGHTED) AND SUBSCALE SCORES

GROUP	N	SES 3 Mean (STD)	FI Mean (STD)	EP Mean (STD)	OE Mean (STD)	SI Mean (STD)	LF Mean (STD)
CONTROL	20	56.5 (13.6)	0.58 (0.61)	0.85 (0.39)	-0.08 (0.46)	0.99 (0.39)	-0.46 (1.25)
UNTREATED	10	53.3 (19.2)	0.73 (0.84)	0.50 (0.82)	-0.03 (0.83)	1.30 (0.36)	0.00 (1.50)
TREATED	65	68.9 (20.8)♦	0.19 (0.19)	0.73 (0.44)	0.40 (0.71)♣	1.16 (0.48)	-0.62 (1.33)

♦ P ≤ 0.05 Compared with Control and Untreated Groups
♣ P ≤ 0.05 Compared with Control Group.
ANOVA OF FI No Significant Difference
ANOVA of EP No Significant Difference
ANOVA of LF No Significant Difference
ANOVA OF SI No Significant Difference
FI Preferred Frequency of Sexual Intercourse
EP Enjoyment Potential of Sexual Interaction.
OE Orgasm Adequacy During Intercourse and its Evaluation
SI Sexual Inhibition
LF Length of Foreplay

Table 27: EFFECTS OF INDEPENDENT VARIABLES ON MALE SES 3 (UNWEIGHTED) AND SES 3 SUBSCALES

INDEPENDENT VARIABLES			
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**
SES 3	β coefficient =- 0.51 P= 0.05	β coefficient = -5.87 P= 0.084	β coefficient = -3.19 P= 0.347
FI	β coefficient = 0.010 P= 0.334	β coefficient = 0.080 P= 0.673	β coefficient =-0.279 P= 0.142
EP	β coefficient = 0.004 P= 0.284	β coefficient = -0.031 P= 0.665	β coefficient = -0.147 P= 0.053
II	β coefficient = 0.01 P= 0.19	β coefficient = 0.05 P= 0.79	β coefficient = 0.22 P= 0.31
LF	β coefficient = 0.04 P= 0.053	β coefficient = -0.18 P= 0.60	β coefficient = -0.33 P= 0.34
OE	β coefficient = 0.01 P= 0.33	β coefficient = -0.11 P= 0.52	β coefficient = -0.06 P= 0.71

FI Preferred Frequency of Sexual Intercourse
EP Enjoyment Potential of Sexual Interaction.
II Inhibition During Intercourse
LF Length of Foreplay
OE Orgasm Adequacy During Intercourse and Its Evaluation

See Statistics Appendix for explanation of dummy variables

Table 28:EFFECTS OF INDEPENDENT VARIABLES ON FEMALE SES 3 (UNWEIGHTED) AND SES 3 SUBSCALES

INDEPENDENT VARIABLES			
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**
SES 3	β coefficient = 0.399 P= 0.122	β coefficient = -9.95 P= 0.05	β coefficient = -1.32 P= 0.768
FI	β coefficient = 0.00 P= 0.595	β coefficient = 0.08 P= 0.664	β coefficient = -0.03 P= 0.811
EP	β coefficient = 0.00 P= 0.593	β coefficient = -0.20 P= 0.078	β coefficient = -0.02 P= 0.744
SI	β coefficient = 0.00 P= 0.75	β coefficient = -0.14 P= 0.175	β coefficient = 0.14 P= 0.171
LF	β coefficient = 0.00 P= 0.957	β coefficient = -0.14 P= 0.609	β coefficient =- 0.35 P= 0.38
OE	β coefficient = 0.00 P= 0.921	β coefficient = 0.05 P= 0.70	β coefficient = 0.00 P= 0.987

FI = Preferred Frequency of Intercourse
EP = Enjoyment Potential of Intercourse
OE = Orgasm Adequacy During Intercourse and its Evaluation
SI = Sexual Inhibition
LF Length of Foreplay

See Statistics Appendix for explanation of dummy variables

RESULTS OF SES 4 ATTRACTION TO MARRIAGE SCALE

There were no significant differences between the three study groups and SES 4 unweighted scores for either sex (Table 29 106 Table 30 106). It should be remembered that on the SES 4 scale a positive score means a positive response unlike the other SES. The male control group had a significantly higher MS (marital satisfaction) score than both the treated and untreated group suggesting that they rated their marriage to be more satisfactory than the other two groups. The control group were also more permissive in their attitudes toward extramarital involvement than the untreated and treated groups(Table 29 106). A similar pattern was seen in the female group with control and untreated groups having a significantly higher mean AE score implying a more permissive attitude to extramarital involvement than the treated group. The female treated group also had a significantly more negative score on the ES (evaluation of partner as sexual partner) subscale than the untreated group suggesting that they found their marriage partner less satisfactory than the untreated group (Table 30 106). It should also be noted that the treated group had a more negative mean score than the control group on the AE subscale implying a more restrictive attitude to extramarital involvement.

Further education, age at time of study and religious affiliation had no predictive value on the male SES 4 unweighted responses (Table 31 107), in the female unweighted SES 4 scale further education exerted a significant effect (Table 32 108). Multiple regression was only performed on the MS and ES subscale of SES 4 in the male data because the AE distribution of scores was not Gaussian. None of the three independent variables mentioned above had a significant effect on these two subscales.

For the women further education had a predictive effect on the AE subscale and age at time of study on the ES subscale.

Table 29:MALE SES 4 (UNWEIGHTED) AND SUBSCALE SCORES

GROUP	N	SES 4 Mean (STD)	MS Mean (STD)	ES Mean (STD)	AE Median (Q1 - Q2)
CONTROL	17	70.2 (6.5)	-0.34 (0.56) *	-0.91(0.26)	0.97 (0.70 - 1.12)
UNTREATED	12	70.8 (12.4)	-0.71 (0.41)	-0.85 (0.21)	0.65 (0.42 - 0.90)
TREATED	73	67.1 (11.2)	-0.62 (0.41)	-0.93 (0.47)	0.58 (0.13 - 0.94)

*** P ≤ 0.05 Compared With Treated and Untreated (ANOVA)**
P = 0.034 for Kruskall-Wallis of AE
ANOVA ES Not significant

Table 30:FEMALE SES 4 (UNWEIGHTED) AND SUBSCALE SCORES

GROUP	N	SES 4 Mean (STD)	MS Mean (STD)	AE Mean (STD)	ES Mean (STD)
CONTROL	20	73.2 (5.23)	-0.62 (0.31)	0.07 (0.50)	-0.47 (0.99)
UNTREATED	10	74.3 (5.37)	-0.62 (0.48)	0.02 (0.77)	-0.10 (1.33)
TREATED	65	68.8 (11.07)	-0.78 (0.50)	-0.43 (0.83) *	-1.04 (1.29) ♣

*** P ≤ 0.05 Compared with the Control Group and Untreated Group**
♣ P ≤ 0.05 Compared with Untreated Group

MS Marital Satisfaction
AE Attitude to Extramarital Involvement
ES Evaluation of Partner as Sexual Partner

Table 31:EFFECTS OF INDEPENDENT VARIABLES ON MALE SES 4 (UNWEIGHTED) AND SES 4 SUBSCALES

INDEPENDENT VARIABLES			
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**
SES4	β coefficient = 0.08 P= 0.49	β coefficient = 0.27 P= 0.90	β coefficient = 2.03 P= 0.35
MS	β coefficient = 0.00 P= 0.42	β coefficient = 0.09 P= 0.38	β coefficient = 0.02 P= 0.82
ES	β coefficient = 0.00 P= 0.51	β coefficient = 0.01 P= 0.86	β coefficient = 0.05 P= 0.60

MS Marital Satisfaction
AE Attitude Towards Extramarital Involvement
ES Evaluation of Partner as Sexual Partner

See Statistics Appendix for explanation of dummy variables

Table 32: EFFECTS OF INDEPENDENT VARIABLES ON FEMALE SES 4 (UNWEIGHTED) AND SES 4 SUBSCALES

INDEPENDENT VARIABLES			
SUBSCALE	**AGE**	**FURTHER EDUCATION**	**RELIGIOUS AFFILIATION**
SES 4	β coefficient = 0.06 P= 0.61	β coefficient = -4.3 P= 0.05	β coefficient = 0.98 P= 0.71
MS	β coefficient = 0.01 P= 0.054	β coefficient =-0.14 P= 0.15	β coefficient =- 0.05 P= 0.86
AE	β coefficient =-0.01 P= 0.11	β coefficient = 0.33 P= 0.05	β coefficient =-0.05 P= 0.79
ES	β coefficient = 0.64 P= 0.05	β coefficient = -0.23 P= 0.38	β coefficient = 0.35 P= 0.31

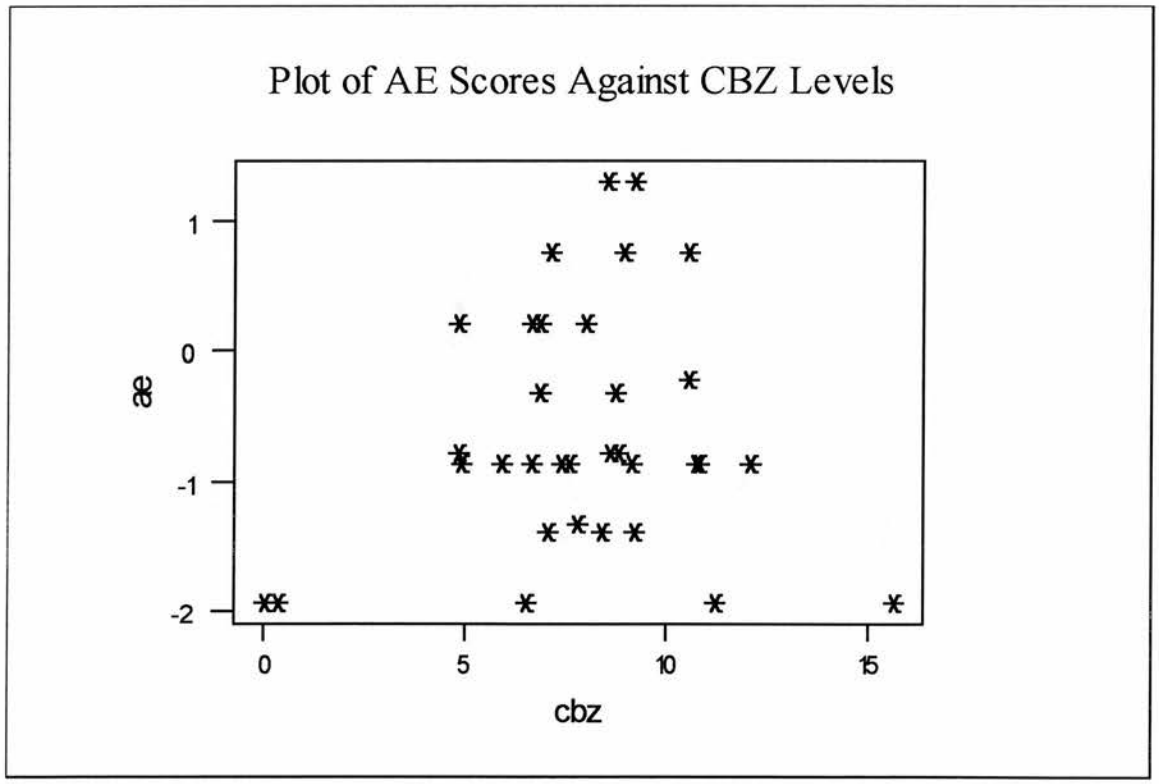
MS Marital Satisfaction
AE Attitude Towards Extramarital Involvement
ES Evaluation of Partner as Sexual Partner

See Statistics Appendix for explanation of dummy variables

EFFECTS OF AED LEVELS ON AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE SCORES

Simple linear regression failed to show any significant relationships between AED levels and AE scores for either men or women. Pearson correlation coefficients were also calculated all of these were of modest values (Table 33 110,Table 34 111,Table 35 112,Table 36 113,Table 37 114,Table 38 115)

Table 33 :LINEAR REGRESSION OF MALE AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE SCORES AGAINST CARBAMAZEPINE LEVELS

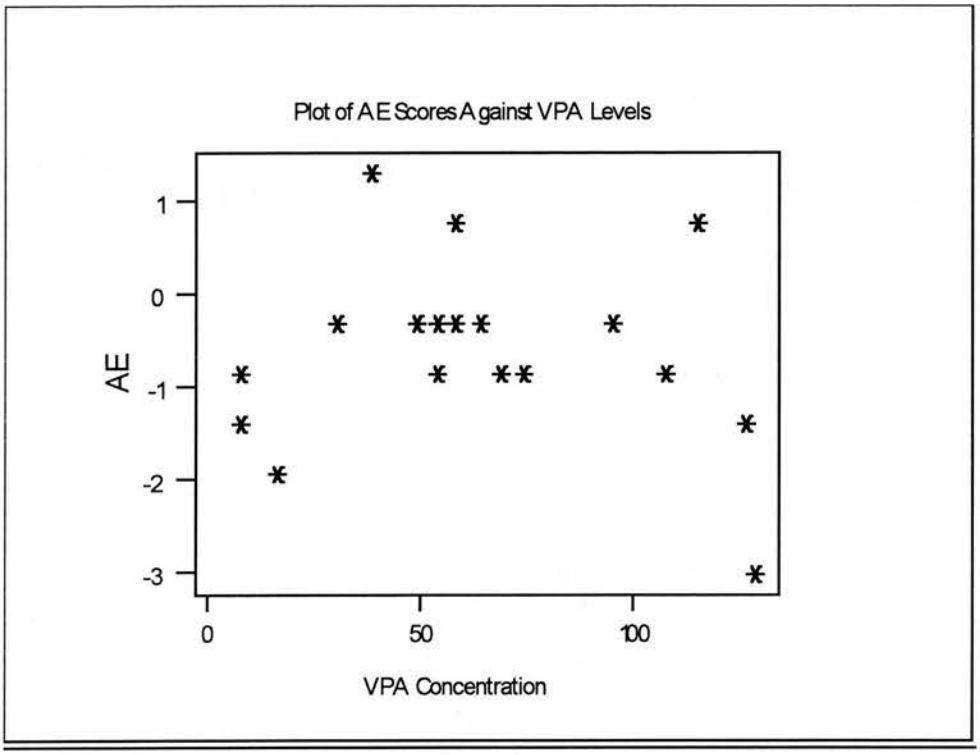


Carbamazepine in μMol/l

Regression Analysis
The regression equation is
AE = - 0.952 + 0.0471 CBZ

Correlation (Pearson) of AE and CBZ = 0.148

Table 34 : LINEAR REGRESSION OF MALE AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE AGAINST SODIUM VALPROATE LEVELS



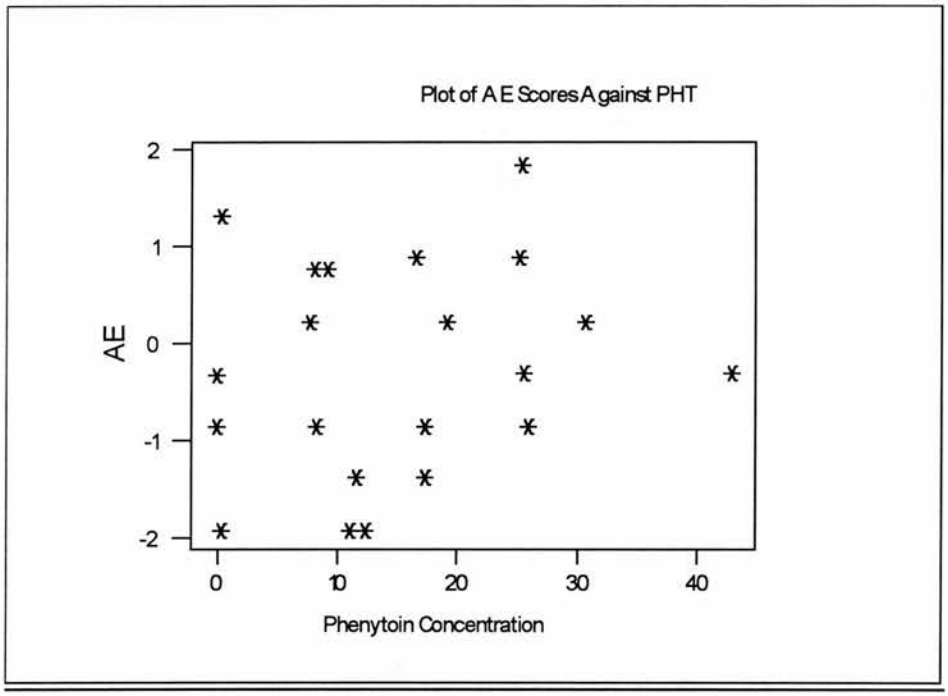
Sodium Valproate in μMol/l

Regression Analysis

The regression equation is
AE = - 0.441 - 0.00381 VPA

Correlation (Pearson) of AE and VPA = -0.121

Table 35:LINEAR REGRESSION OF MALE AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE AGAINST PHENYTOIN LEVELS



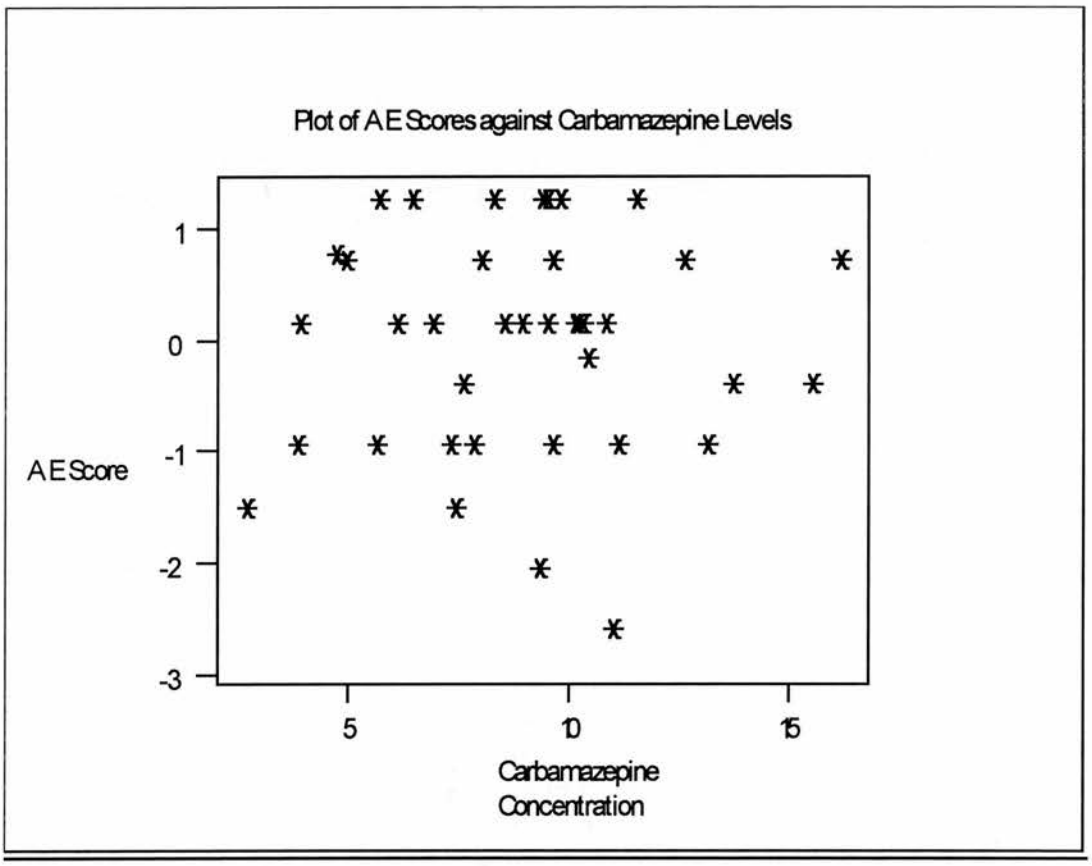
Phenytoin in μMol/l

The regression equation is

AE = - 0.577 + 0.0233 PHT

Correlation (Pearson) = AE and PHT 0.135

Table 36: LINEAR REGRESSION OF FEMALE AE (AROUSAL BY EROTIC IMAGERY) AGAINST CARBAMAZEPINE LEVELS

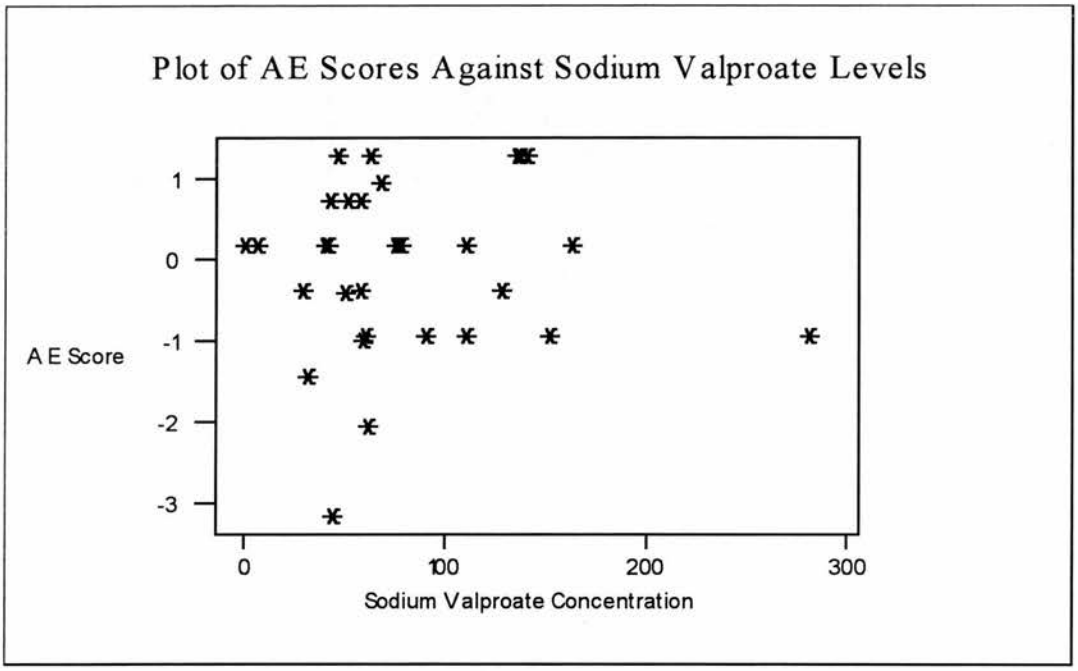


Carbamazepine in µMol/l

The regression equation is
AE = - 0.172 + 0.0153 CBZ Level

Correlation (Pearson) of AE and CBZ Levels = 0.047

Table 37: LINEAR REGRESSION OF FEMALE AE (AROUSAL BY EROTIC IMAGERY) SCORES AGAINST SODIUM VALPROATE LEVELS

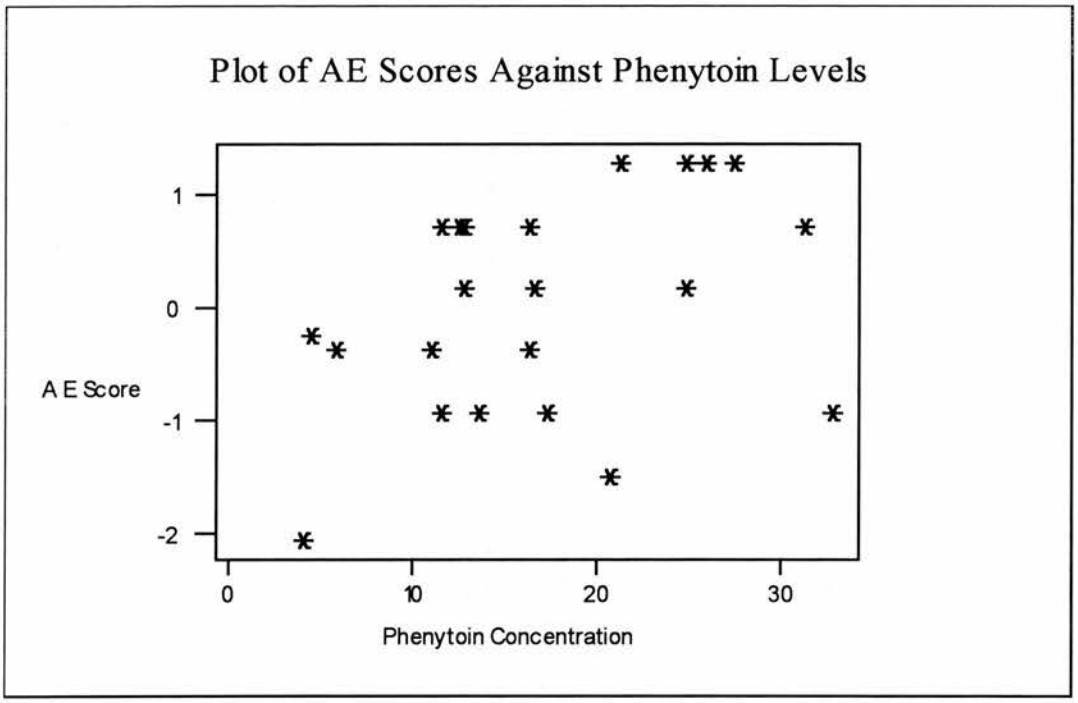


Sodium Valproate in μMol/l

The regression equation is
AE = - 0.169 + 0.00049 VPA Level

Correlation of AE and VPA = 0.025

Table 38: LINEAR REGRESSION OF FEMALE AE (AROUSAL BY EROTIC IMAGERY) AGAINST PHENYTOIN LEVELS



Phenytoin in μMol/l

The regression equation is
AE = - 0.757 + 0.0455 PHT level

Correlation (Pearson) of AE and PHT = 0.386

EFFECTS OF SEIZURE TYPE, FREQUENCY AND AGE AT ONSET ON SES SCORES

Subjects were divided into three seizure type group as described in the methods section. For SES 1 both the male CPS and PGE groups had significantly higher MS mean subscale scores than the controls implying that they espoused a stricter sexual morality within marriage (Table 39 117), an identical pattern was seen for the female group Table 40 118.

Table 39: MALE SES 1 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE

GROUP	N	MS Mean (STD)	PS Mean (STD)	PA Mean (STD)
Complex Partial Seizures	75	0.23 (0.57)*	-0.41 (0.83)	-0.07 (0.42)
Primary Generalised Epilepsy	86	0.10 (0.44)*	-0.45 (1.00)	-0.02 (0.34)
Undefined	26	0.09 (0.62)	-0.42 (0.77)	0.07 (0.52)
Control	45	0.17 (0.40)	-0.46 (0.99)	-0.03 (0.34)

ANOVA MS * Denotes $P \leq 0.05$ Compared with Control Group
ANOVA PS P = 0.993
ANOVA PA P = 0.620

MS Sexual Morality Within Marriage
PS Premarital Sexual Experience and Attitude
PA Premarital Sexual Attitude

Table 40: **FEMALE SES 1 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE**

GROUP	N	MS Mean (STD)	PS Mean (STD)	SS Mean STD)
Complex Partial Seizures	110	0.16 (0.62)*	-0.09 (0.80)	-0.04 (0.65)
Primary Generalised Epilepsy	56	0.04 (0.50) *	-0.11 (0.69)	-0.04 (0.62)
Undefined	28	0.06 (0.53)	-0.31 (0.66)	-0.07 (0.65)
Control	48	-0.18 (0.54)	-0.35 (0.69)	-0.31 (0.53)

ANOVA MS * $P \leq 0.05$ Compared with the Control Group
ANOVA PS $P = 0.154$
ANOVA SS $P = 0.095$

MS Sexual Morality Within Marriage
PS Premarital Sexual Experience and Attitude
SS Sexual Socialisation Morality

There were no significant differences in the male IS or EE subscales of SES 2, but the PGE and undefined groups had significantly higher mean AE scores than the control group suggesting they were less aroused by erotic imagery(Table 41 119).

Table 41: MALE SES 2 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE

GROUP	N	IS Mean (STD)	EE Mean (STD)	AE Mean (STD)
Complex Partial Seizures	75	-0.46 (0.67)	-0.25 (0.68)	-0.56 (1.09)
Primary Generalised Epilepsy	86	-0.57 (0.79)	-0.25 (0.80)	-0.47 (1.12)*
Undefined	26	-0.63 (0.80)	-0.25 (1.10)	-0.11 (1.06)*
Control	45	-0.83 (0.55)	-0.38 (0.62)	-0.92 (0.96)

IS Interpersonal Sexual Attraction P = 0.096 (ANOVA)
EE Evaluation of Exposure to Erotic Imagery P = 0.815 (ANOVA)
AE Arousal By Erotic Imagery
*** Denotes $P \leq 0.05$ Compared with Control (ANOVA)**

The female CPS and undefined groups had significantly higher mean IS scores compared with controls implying lower interpersonal sexual attraction, and the CPS group had a significantly higher mean EE score compared with controls and PGE group suggesting they did not enjoy exposure to erotic imagery as much as the other two groups (Table 42 120)

Table 42: **FEMALE SES 2 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE**

GROUP	N	IS Mean (STD)	EE Mean (STD)	AE Mean (STD)
Complex Partial Seizures	110	-0.19 (0.78)*	0.32 (0.73)	-0.01 (1.0)
Primary Generalised Epilepsy	56	-0.42 (0.84)	0.02 (0.91) ♣	-0.29 (1.1)
Undefined	28	-0.23 (0.75) *	0.12 (0.72)	-0.21 (1.1)
Control	48	-0.66 (0.57)	-0.18 (0.78)♣	-0.35 (0.95)

*** P ≤ 0.05 Compared with Control Group**
♣ P ≤ 0.05 Compared with Complex Partial Seizure Group
ANOVA of AE Not Significant (P = 0.224)

IS Interpersonal Sexual Attraction
EE Evaluation of Exposure to Erotic Imagery
AE Arousal By Erotic Imagery

There were no significant differences between the seizure type groups and any of the SES 3 subscales for either men or women Table 43 121 Table 44 122).

Table 43: MALE SES 3 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE

GROUP	N	FI Mean (STD)	EP Mean (STD)	LF Mean (STD)	OE Mean (STD)	II Mean (STD)
Complex Partial Seizures	40	0.16 (0.86)	0.91 (0.43)	-0.29 (1.15)	1.82 (0.82)	1.59 (0.89)
Primary Generalised Epilepsy	38	0.39 (0.78)	0.86 (0.33)	-0.41 (1.81)	1.68 (0.75)	1.35 (0.90)
Undefined	12	0.24 (1.02)	0.92 (0.26)	-0.15 (1.48)	1.70 (0.69)	1.44 (0.89)
Control	17	0.23 (0.76)	0.97 (0.20)	-0.32 (1.17)	1.68 (0.73)	1.28 (0.78)

ANOVA OF Subscales Shows No Significant Differences

FI Preferred Frequency of Sexual Intercourse
EP Enjoyment Potential of Sexual Interaction.
OE Orgasm Adequacy During Intercourse and its Evaluation
SI Sexual Inhibition
LF Length of Foreplay

Table 44: FEMALE SES 3 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE

GROUP	N	FI Mean (STD)	EP Mean (STD)	SI Mean (STD)	OE Mean (STD)	LF Mean (STD)
Complex Partial Seizures	43	0.23 (1.02)	0.66 (0.60)	1.17 (0.47)	0.21 (0.79)	-0.76 (1.34)
Primary Generalised Epilepsy	16	0.22 (0.69)	0.79 (0.42)	1.13 (0.48)	0.31 (0.78)	-0.72 (1.22)
Undefined	15	0.32 (0.72)	0.76 (0.37)	1.30 (0.45)	0.59 (0.52)	0.04 (1.32)
Control	20	0.59 (0.62)	0.84 (0.40)	0.97 (0.38)	-0.05 (0.45)	-0.50 (1.26)

ANOVA OF Subscales Shows No Significant Differences

FI Preferred Frequency of Sexual Intercourse
EP Enjoyment Potential of Sexual Interaction.
OE Orgasm Adequacy During Intercourse and its Evaluation
SI Sexual Inhibition
LF Length of Foreplay

There were no significant differences between seizure type groups on SES 4 subscale for either sex (Table 45 123, Table 46 123)

Table 45: MALE SES 4 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE

GROUP	N	MS Mean (STD)	ES Mean (STD)		AE Median (Q1 - Q2)
Complex Partial Seizures	40	-0.68 (0.52)	-1.01(0.39)		-0.97 (-0.95 -0.41)
Primary Generalised Epilepsy	38	-0.63 (0.26)	-0.87 (0.45)		-0.88 (-0.79 -0.48)
Undefined	12	-0.56 (0.08)	-0.85 (0.43)		-0.89 (-0.62 -0.47)
Control	17	-0.34 (0.56)	-0.91 (0.26)		-0.92 (-0.65 -0.27)

No Significant Differences (MS and ES by ANOVA, AE by Kruskal-Wallis)

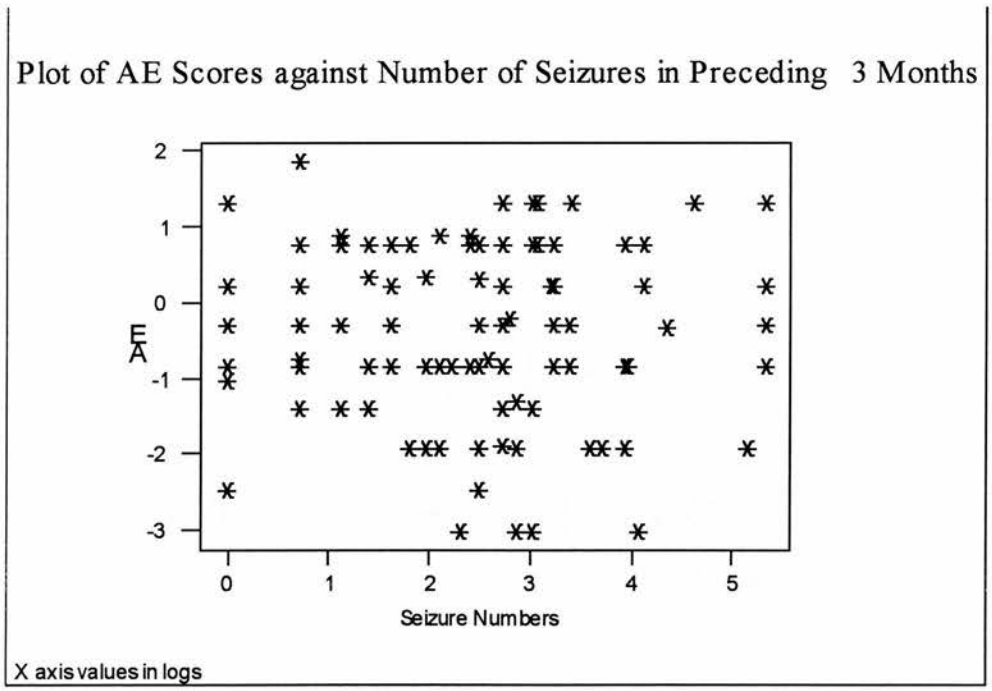
Table 46: FEMALE SES 4 SUBSCALES (WEIGHTED) FOR SEIZURE TYPE

GROUP	N	MS Mean (STD)	AE Mean (STD)	ES Mean (STD)
Complex Partial Seizures	43	-0.73 (0.46)	-0.34 (0.83)	-0.62 (1.15)
Primary Generalised Epilepsy	16	-0.76 (0.59)	-0.39 (0.78)	-1.11 (1.40)
Undefined	15	-0.83 (0.47)	-0.45 (0.93)	-1.43 (1.40)
Control	20	-0.65 (0.30)	0.15 (0.45)	-0.42 (1.00)

MS Marital Satisfaction
AE Attitude to Extramarital Involvement
ES Evaluation of Partner as Sexual Partner

In the male group simple linear regression of number of seizures in the preceding three months and AE (arousal by erotic imagery)(Table 47 124) scores and FI (preferred frequency of intercourse)(Table 48,125) scores did not show any significant relationship, Pearson correlation coefficients were modest. There was no significant relationship between age at onset of epilepsy and AE scores (Table 49,126).

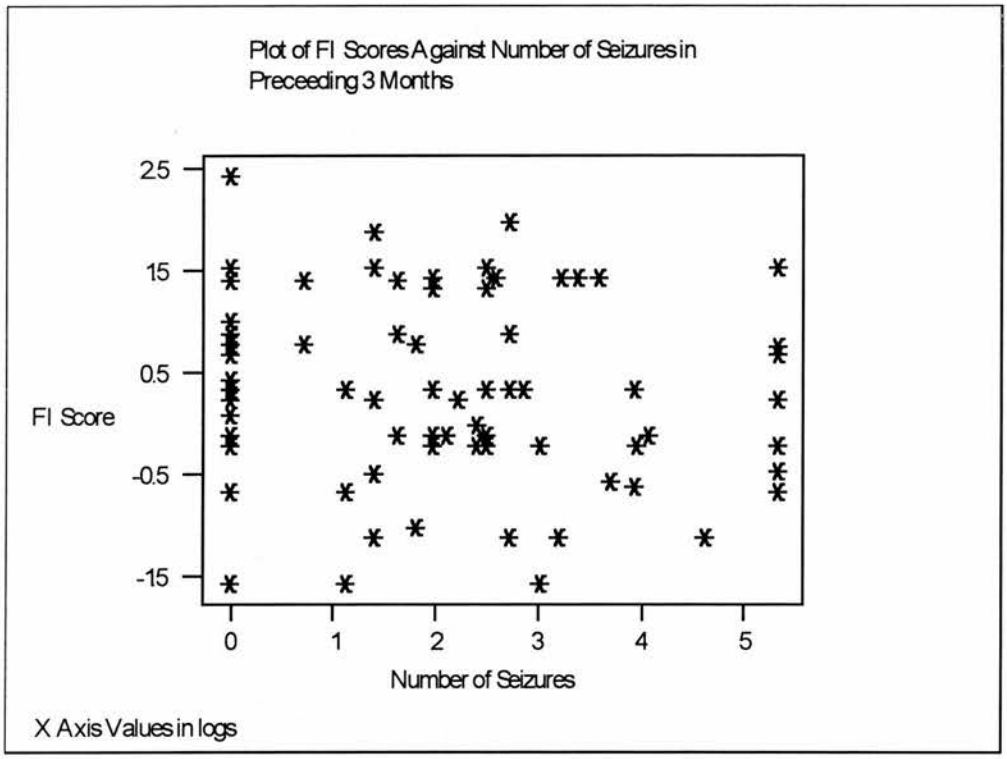
Table 47: LINEAR REGRESSION OF NUMBER OF SEIZURES IN PRECEDING THREE MONTHS AND AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE (MEN)



The regression equation is
AE = - 0.546 + 0.024 SEIZURES

Correlation of (Pearson) AE and SEIZURES = 0.029

Table 48: LINEAR REGRESSION OF NUMBER OF SEIZURES IN PRECEDING THREE MONTHS AND FI (PREFERRED FREQUENCY OF INTERCOURSE) SUBSCALE (MEN)



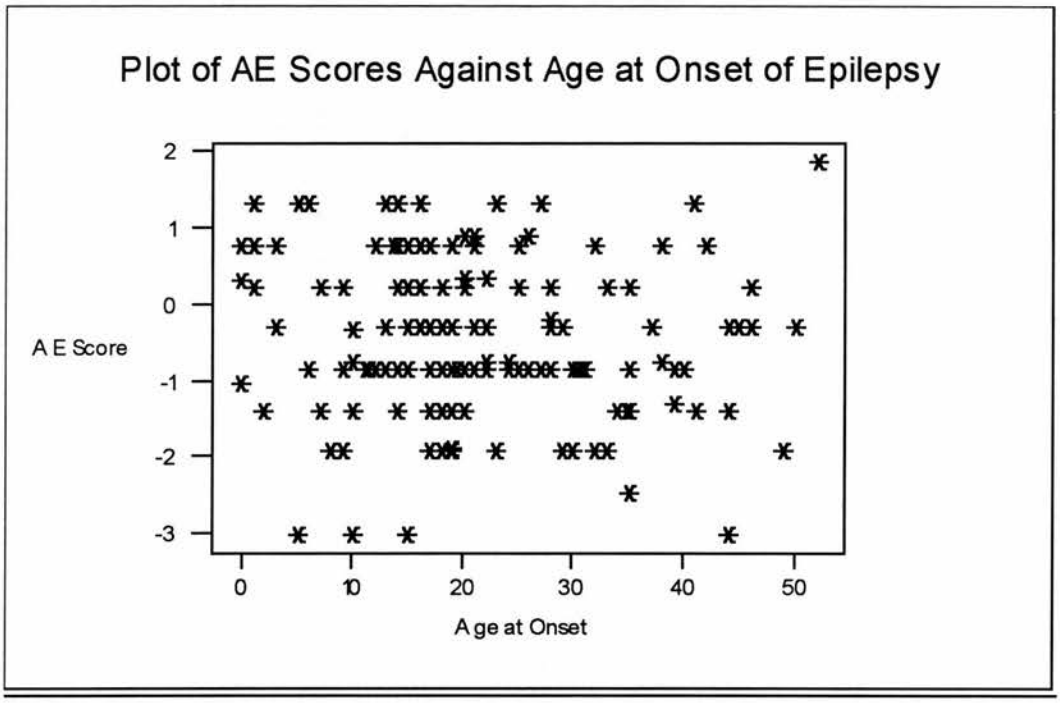
Regression Analysis(seizure numbers transformed to logs see statistical appendix)

The regression equation is
FI = 0.581 - 0.108 SEIZURES

Correlation (Pearson) of FI and SEIZURES = -0.187

(49 of the subjects had not had any seizures in the preceeding 3 months. When logarithmic transformation performed Minitab registers these as 0 for analytical purposes, but the graphics package registers them as missing values for the purpose of plotting a scatter graph. Thus there appear to be fewer values on the graph than are used in the calculation.)

Table 49: LINEAR REGRESSION OF AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE SCORE AND AGE AT ONSET OF EPILEPSY (MEN)



The regression equation is
AE = - 0.204 - 0.0109 Age at Onset

Correlation of AE and Age at onset = -0.124

Spearman rank correlation of number of seizures in previous 3 months and AE scores for women did not show any significant relationship (Table 50,127). Unlike the men the distribution of seizure numbers was not Gaussian and could not be rendered so by logarithmic transformation. There was a significant relationship between FI scores and seizure number with the scatterplot suggesting that the more frequent the seizures the higher the FI score ie the individual was less likely to want intercourse (Table 51,128). There was no significant relationship between AE scores and age at onset of epilepsy (Table 52,129).

Table 50:CORRELATION OF SEIZURE FREQUENCY AND AE SCORES (WOMEN)

Correlation of Seizure Frequency and AE Score = 0.022

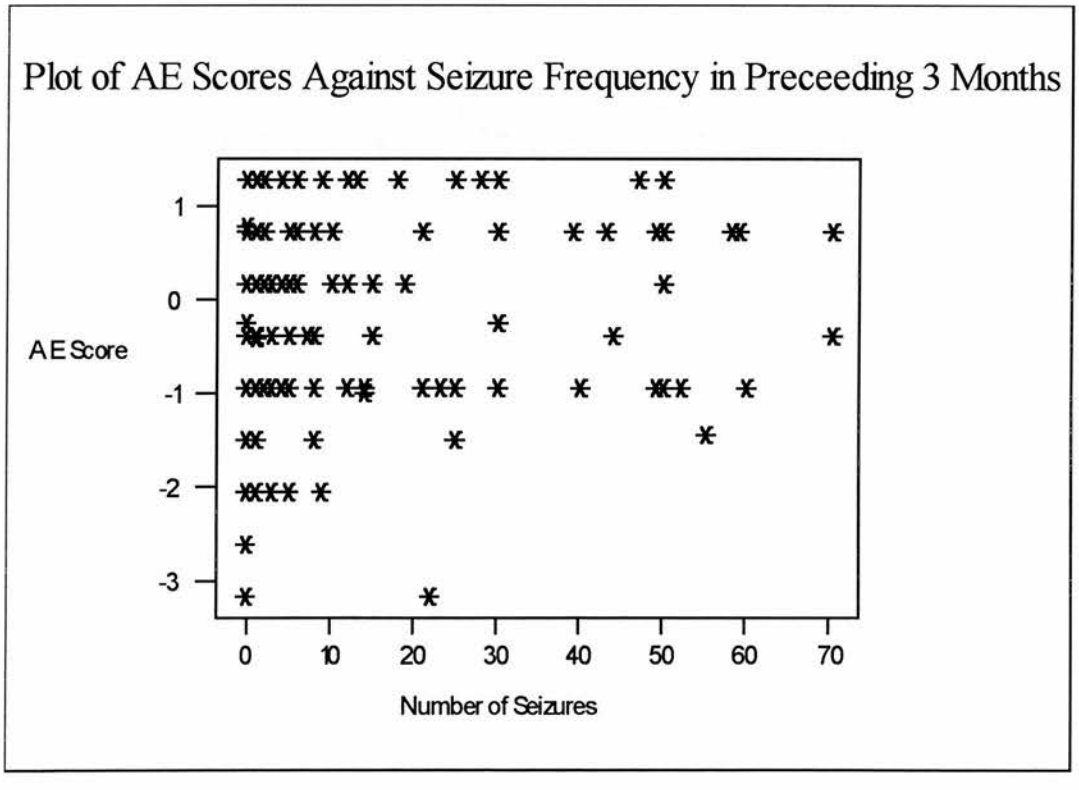
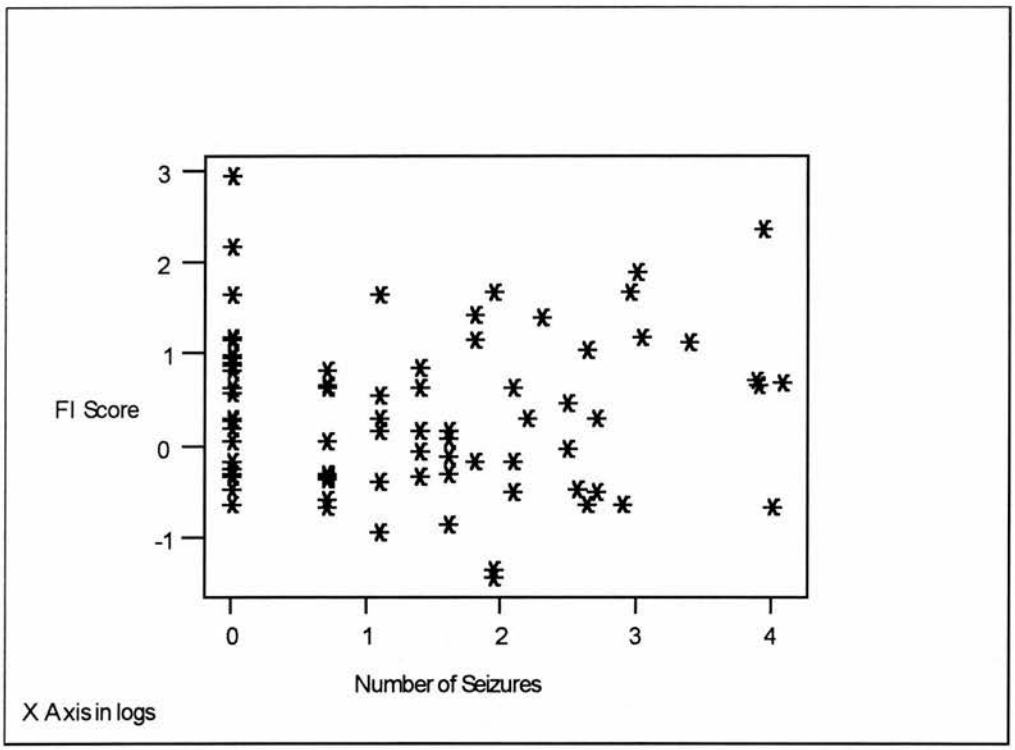


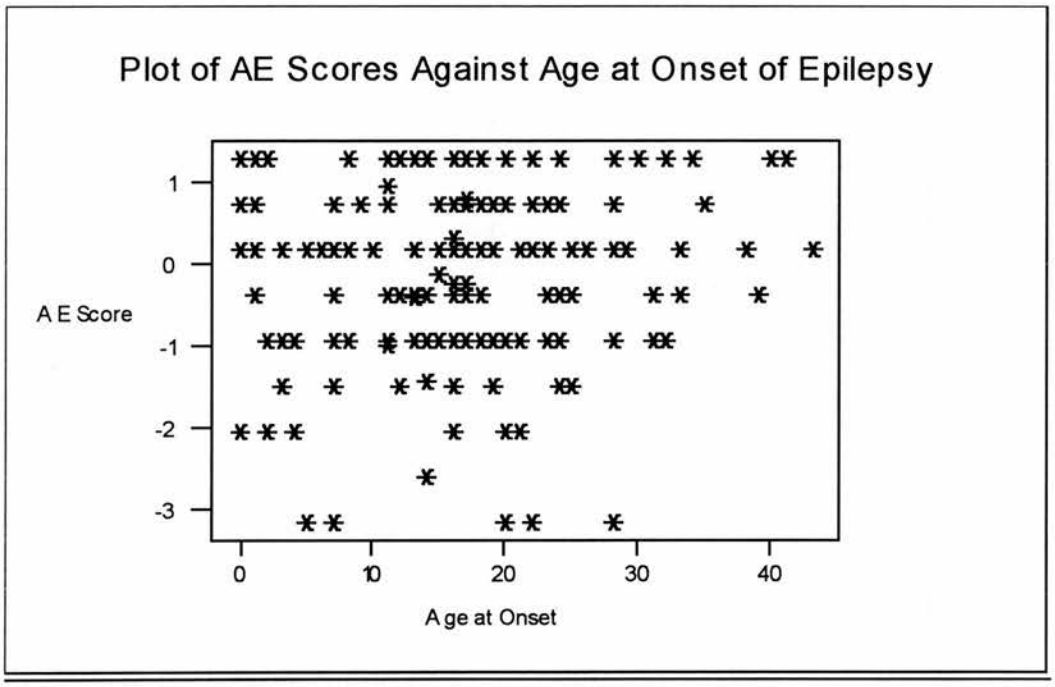
Table 51: LINEAR REGRESSION OF FI SUBSCALE SCORES AND SEIZURE FREQUENCY IN PRECEDING 3 MONTHS (WOMEN)



The regression equation is
FI = - 0.285 + 0.252 Number of Seizures (logs)

Correlation (Pearson) of FI and Number of Seizures (logs) = 0.376

Table 52: LINEAR REGRESSION OF AGE AT ONSET OF EPILEPSY AN AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE SCORES (WOMEN)



The regression equation is
AE = - 0.282 + 0.0103 ageon

Correlation of AE and age at onset of epilepsy = 0.085

EFFECTS OF ANTIEPILEPTIC DRUGS ON HORMONE LEVELS AND SEX HORMONE BINDING GLOBULIN.

The male CBZ and PHT monotherapy groups and polytherapy group had significantly higher mean SHBG concentrations than the control and untreated groups, in addition the PHT and polytherapy groups had higher mean SHBG levels than the VPA group(Table 53,131)

Table 53: MALE SEX HORMONE BINDING GLOBULIN CONCENTRATION

GROUP	N	MEAN (μMol/L)	STANDARD DEVIATION
CONTROL	33	26.1	11.9
UNTREATED	32	32.8	12.8
SODIUM VALPROATE	18	35.5	17.6
CARBAMAZEPINE	31	49.1* •	30.1
PHENYTOIN	21	56.2* • ♣	30.7
POLYTHERAPY	48	58.4* • ♣	30.9

*** P ≤ 0.05 Compared with Control Group (ANOVA)**
• P ≤ 0.05 Compared with Untreated Group (ANOVA)
♣P ≤ 0.05 Compared with Sodium Valproate Group (ANOVA)

The PHT and polytherapy groups had significantly higher mean total testosterone levels than the control, VPA and CBZ groups, in addition the PHT group had a significantly higher mean total testosterone than the untreated group (Table 54,132). There were no significant differences between the six groups when free testosterone was calculated, and all the group means fell well within the Royal Infirmary of Glasgow's normal range (Table 55,132).

Table 54:MALE TOTAL TESTOSTERONE CONCENTRATION

GROUP	N	MEAN (ηMol/L)	STANDARD DEVIATION
CONTROL	33	16.6	5.0
UNTREATED	32	19.3	5.0
SODIUM VALPROATE	18	16.2	6.6
CARBAMAZEPINE	31	18.7	5.7
PHENYTOIN	21	24.1* • ♣ ♠	9.0
POLYTHERAPY	48	22.2* ♣ ♠	7.7

*** P ≤ 0.05 Compared with Control Group (ANOVA)**
• P ≤ 0.05 Compared with Untreated Group (ANOVA)
♣ P ≤ 0.05 Compared with Sodium Valproate Group (ANOVA)
♠ P ≤ 0.05 Compared with Carbamazepine Group (ANOVA)

Table 55: MALE CALCULATED FREE TESTOSTERONE CONCENTRATION

GROUP	N	MEAN (ρMol/L)	STANDARD DEVIATION
CONTROL	33	467.7	150.6
UNTREATED	32	488.6	111.7
SODIUM VALPROATE	18	399.9	140.0
CARBAMAZEPINE	31	408.2	130.1
PHENYTOIN	21	471.4	130.6
POLYTHERAPY	48	430.9	141.6

ANOVA P = 0.093 (ANOVA)

The CBZ, PHT and polytherapy groups had significantly lower dehydroepiandosterone sulphate (DHEAS) levels than the controls, untreated and VPA groups, in addition the

polytherapy group had a significantly lower mean DHEAS concentration than the CBZ. group (Table 56,133). ANOVA of androstenedione levels showed that the PHT monotherapy groups had a significantly higher mean concentration than the controls, untreated, VPA or CBZ groups (Table 57,133).

Table 56 :MALE DEHYDROEPIANDOSTERONE SULPHATE CONCENTRATIONS

GROUP	N	MEAN (μmol/L)	STANDARD DEVIATION
CONTROL	33	9.01	6.03
UNTREATED	32	9.40	5.04
SODIUM VALPROATE	18	10.28	5.37
CARBAMAZEPINE	31	5.62 * ♣ ♦	3.45
PHENYTOIN	21	4.42 * ♣ ♦	3.51
POLYTHERAPY	48	3.30 * ♣ ♦ ♥	2.13

*** P ≤ 0.05 Compared with Control Group (ANOVA)**
♣ P ≤ 0.05 Compared with Untreated Group (ANOVA)
♦ P ≤ 0.05 Compared with Sodium Valproate Group (ANOVA)
♥ P ≤ 0.05 Compared with Carbamazepine Group (ANOVA)

Table 57 : MALE ANDROSTENEDIONE CONCENTRATIONS

GROUP	N	MEAN (ηmol/L)	STANDARD DEVIATION
CONTROL	33	6.20	2.33
UNTREATED	32	6.05	1.50
SODIUM VALPROATE	18	5.90	2.03
CARBAMAZEPINE	31	6.10	2.14
PHENYTOIN	21	8.12 *	3.06
POLYTHERAPY	48	6.90	2.80

*** P ≤ 0.05 Compared with Control, Untreated, Sodium Valproate, and Carbamazepine Groups (ANOVA)**

Because of the small numbers involved it was not possible to analyse female hormone results in drug treatment groups as was done for the male data. The women were divided into those receiving treatment and those who were not. Thus the untreated group was a mixture of healthy controls and people with epilepsy.

ANOVA of SHBG for both treated and untreated groups showed no significant change in concentrations during the menstrual cycle (Table 58,134,Table 59,134)

Table 58: FEMALE UNTREATED SHBG CONCENTRATIONS

GROUP	N	MEAN (μmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	18	50.44	14.88
Ovulatory (days 11-17 of menstrual cycle)	11	47.70	30.12
Luteal (days 20-28 of menstrual cycle)	14	74.50	44.66

P = 0.059 (ANOVA)

Table 59:FEMALE TREATED GROUP SHBG CONCENTRATIONS

GROUP	N	MEAN (μmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	27	122.26	48.97
Ovulatory (days 11-17 of menstrual cycle)	30	90.83	49.68
Luteal (days 20-28 of menstrual cycle)	31	112.89	63.48

P = 0.105 (ANOVA)

Comparison of treated and untreated groups' SHBG for proliferative, ovulatory and luteal phases of the menstrual cycle showed that those women had significantly higher mean SHBG levels than those women not receiving AEDS (Table 60,135,Table 61,Table 62,135)

Table 60: FEMALE UNTREATED AND TREATED GROUPS PROLIFERATIVE PHASE SHBG CONCENTRATIONS

GROUP	N	MEAN (μmol/L)	STANDARD DEVIATION
Untreated	18	50.44	14.88
Treated	27	122.3	49.0

P ≤ 0.00(Two sample T)

Table 61:FEMALE UNTREATED AND TREATED OVULATORY PHASE SHBG CONCENTRATIONS

GROUP	N	MEAN (μmol/L)	STANDARD DEVIATION
Untreated	11	47.70	30.12
Treated	30	79.80	49.00

P = 0.016(Two sample T)

Table 62: FEMALE TREATED AND UNTREATED LUTEAL PHASE SHBG CONCENTRATIONS

GROUP	N	MEAN (μmol/L)	STANDARD DEVIATION
Untreated	14	74.5	44.7
Treated	31	112.9	63.5

P = 0.031(Two sample T)

Comparison of total testosterone levels for the proliferative, ovulatory, and luteal phases of the menstrual cycle showed there were no significant differences between the three phases in either the treated and untreated women (Table 63,137,Table 64,137).

Table 63:FEMALE UNTREATED TOTAL TESTOSTERONE CONCENTRATIONS

GROUP	N	MEAN (nmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	18	1.41	0.56
Ovulatory (days 11-17 of menstrual cycle)	11	1.70	0.50
Luteal (days 20-28 of menstrual cycle)	14	1.75	1.14

P = 0.262(ANOVA)

Table 64:FEMALE TREATED TOTAL TESTOSTERONE CONCENTRATIONS

GROUP	N	MEAN (nmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	27	3.52	1.26
Ovulatory (days 11-17 of menstrual cycle)	30	3.06	2.36
Luteal (days 20-28 of menstrual cycle)	31	2.51	1.91

P = 0.699 (ANOVA)

Comparison of treated and untreated groups' total testosterone for proliferative, and ovulatory phases of the menstrual cycle showed that the treated women had significantly higher mean total testosterone than the untreated group (Table 65,137,Table

66,137). There was no significant difference between treated and untreated women in the luteal phase(Table 67,137).

Table 65:FEMALE TREATED AND UNTREATED PROLIFERATIVE TOTAL TESTOSTERONE

GROUP	N	MEAN (nmol/L)	STANDARD DEVIATION
Untreated	18	1.41	0.56
Treated	27	3.53	1.27

P = 0.002(Two sample T)

Table 66:FEMALE TREATED AND UNTREATED OVULATORY TOTAL TESTOSTERONE

GROUP	N	MEAN (nmol/L)	STANDARD DEVIATION
Untreated	11	1.70	1.14
Treated	30	3.07	2.36

P = 0.02(Two sample T)

Table 67:FEMALE TREATED AND UNTREATED LUTEAL TOTAL TESTOSTERONE

GROUP	N	MEAN (nmol/L)	STANDARD DEVIATION
Untreated	14	1.75	0.50
Treated	31	2.51	1.91

P = 0.09(Two sample T)

ANOVA of calculated free testosterone showed no significant differences between proliferative,ovulatory and luteal phases of the untreated group (Table 68,138). There was a significant difference in mean ovulatory free testosterone compared with both proliferative and luteal phases in the treated group (Table 69,138). But no significant differeneces between FT for treated and untreated across the menstrual cycle

Table 68:FEMALE UNTREATED FREE TESTOSTERONE CONCENTRATIONS

GROUP	N	MEAN (pmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	18	29.84	12.29
Ovulatory (days 11-17 of menstrual cycle)	11	33.00	13.17
Luteal (days 20-28 of menstrual cycle)	14	37.54	29.43

P = 0.555 (ANOVA)

Table 69:FEMALE TREATED FREE TESTOSTERONE CONCENTRATIONS

GROUP	N	MEAN (pmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	27	29.99	18.47
Ovulatory (days 11-17 of menstrual cycle)	30	42.46	23.15 *
Luteal (days 20-28 of menstrual cycle)	31	30.08	15.24

*** P ≤ 0.05 Compared with Proliferative and Luteal Groups(ANOVA)**

Table 92: COMPARISON OF UNTREATED AND TREATED PROLIFERATIVE, OVULATORY AND LUTEAL FREE TESTOSTERONE

GROUP		N	MEAN (pmol/L)	STANDARD DEVIATION	Two Sample T
Proliferative phase	Untreated	18	29.84	12.29	P= 0.97
	Treated	27	29.99	18.47	
Ovulatory phase	Untreated	11	33.00	13.17	P=0.64
	Treated	30	42.46	23.15	
Luteal	Untreated	14	37.54	29.43	P = 0.67
	Treated	31	30.08	15.24	

No significant differences between Untreated and Treated Proliferative, Ovulatory, and Luteal Free Testosterone (Two Sample T)

There were no significant differences in mean DHEAS levels between the proliferative, ovulatory, or luteal groups whether receiving AED therapy or not (Table 70,139,Table 71,139)

Table 70: FEMALE UNTREATED DHEAS CONCENTRATIONS

GROUP	N	MEDIAN (μmol/L)	Q1	Q3
Proliferative (days 1-7 of menstrual cycle)	18	3.80	2.75	9.15
Ovulatory (days 11-17 of menstrual cycle)	11	5.30	2.80	11.60
Luteal (days 20-28 of menstrual cycle)	14	6.65	3.95	8.65

P = 0.516(Kruskal-Wallis)

Table 71:FEMALE TREATED DHEAS CONCENTRATIONS

GROUP	N	MEDIAN (μmol/L)	Q1	Q3
Proliferative (days 1-7 of menstrual cycle)	27	2.00	2.00	4.87
Ovulatory (days 11-17 of menstrual cycle)	30	2.60	2.00	4.53
Luteal (days 20-28 of menstrual cycle)	31	2.80	2.00	5.80

P = 0.877(Kruskal-Wallis)

Comparison of the untreated and treated groups' proliferative, ovulatory and luteal phase DHEAS showed that in each case the mean concentration of the treated group was significantly lower than that of the untreated group (Table 72,140,Table 73,140,Table 74,140)

Table 72: FEMALE TREATED AND UNTREATED PROLIFERATIVE DHEAS

GROUP	N	MEDIAN (ηmol/L)	Q1	Q3
Untreated	18	3.80	2.75	9.15
Treated	27	2.10	2.00	4.87

P = 0.04(Mann-Whitney)

Table 73:FEMALE TREATED AND UNTREATED OVULATORY DHEAS

GROUP	N	MEDIAN (ηmol/L)	Q1	Q3
Untreated	11	5.30	2.80	11.60
Treated	30	2.20	2.00	4.53

P = 0.025(Mann-Whitney)

Table 74: FEMALE TREATED AND UNTREATED LUTEAL DHEAS

GROUP	N	MEDIAN (ηmol/L)	Q1	Q3
Untreated	14	6.65	3.95	8.65
Treated	31	2.80	2.00	5.80

P = 0.015(Mann-Whitney)

The mean ovulatory phase androstenedione was significantly higher than that of the proliferative phase(Table 75,141). There was no significant difference noted in the treated group, although mean androstenedione levels were higher in the ovulatory phase of the cycle than in both the proliferative and luteal (Table 76,141).

Table 75: FEMALE TREATED ANDROSTENEDIONE CONCENTRATIONS

GROUP	N	MEAN (ηmol/L)	STANDARD DEVIATION
Proliferative (days 1-7 of menstrual cycle)	18	4.45	1.90
Ovulatory (days 11-17 of menstrual cycle)	11	7.07 *	3.22
Luteal (days 20-28 of menstrual cycle)	14	5.66	1.69

*** P = 0.02 Compared with Proliferative Phase(ANOVA)**

Table 76:FEMALE TREATED ANDROSTENEDIONE CONCENTRATIONS

GROUP	N	MEAN (ηmol/L)	STANDARD DEVIATION
Proliferative	27	6.47	2.90
Mid- Cycle	30	7.70	4.31
Luteal	31	6.67	3.71

P = 0.404(ANOVA)

Comparison of treated and untreated groups androstenedione showed that the treated group had a significantly higher mean proliferative phase concentration than the untreated group (Table 77,142). Otherwise there were no significant differences (Table 78,142 Table 79,142)

Table 77: FEMALE TREATED AND UNTREATED PROLIFERATIVE ANDROSTENEDIONE

GROUP	N	MEDIAN (ηmol/L)	Q1	Q3
Untreated	18	4.2	3.15	5.72
Treated	27	6.0	4.40	8.40

P = 0.021(Mann-Whitney)

Table 78: FEMALE TREATED AND UNTREATED OVULATORY ANDROSTENEDIONE

GROUP	N	MEDIAN (ηmol/L)	Q1	Q3
Untreated	11	7.20	5.40	8.00
Treated	30	6.15	4.80	8.45

P = 0.59(Mann-Whitney)

Table 79:FEMALE TREATED AND UNTREATED LUTEAL ANDROSTENEDIONE

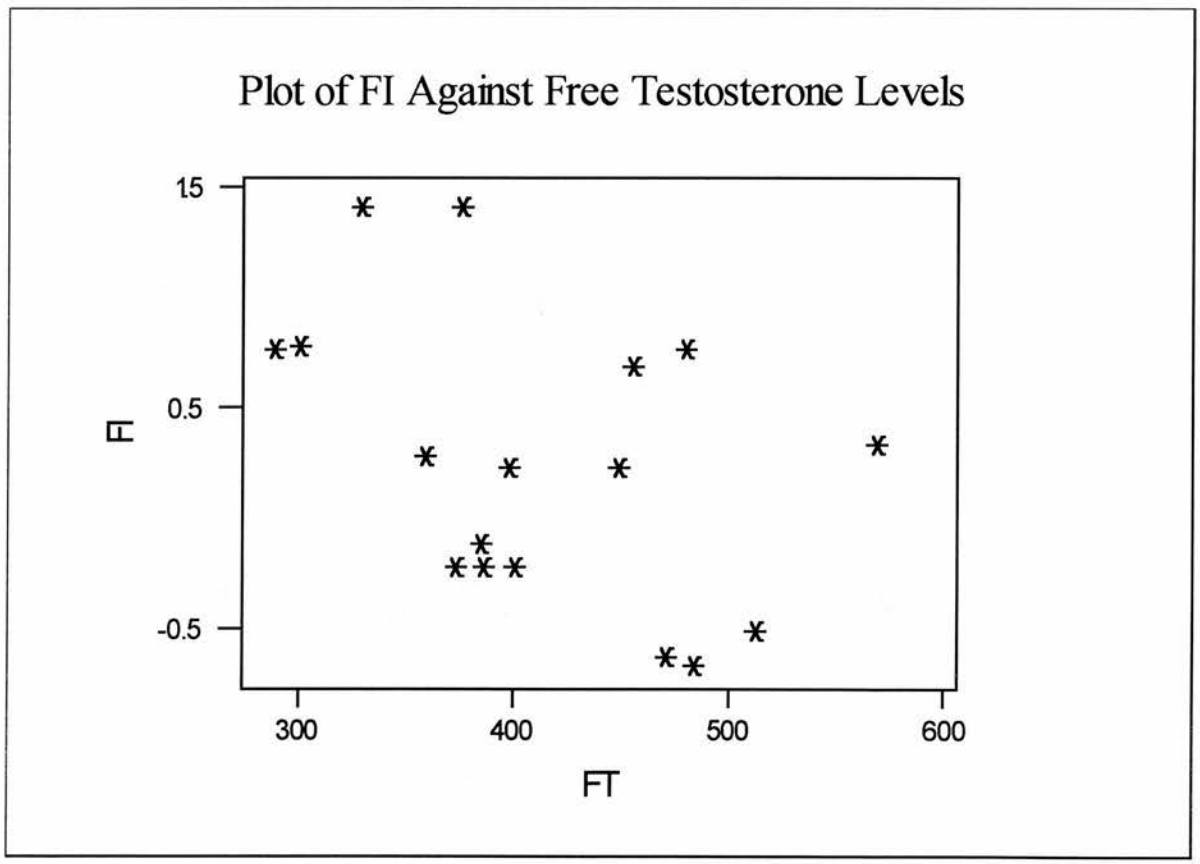
GROUP	N	MEDIAN (ηmol/L)	Q1	Q3
Untreated	14	5.70	4.25	7.15
Treated	31	5.60	3.80	8.40

P = 0.73

RESULTS OF LINEAR REGRESSION OF FI AND EP SCORES AND FREE TESTOSTERONE CONCENTRATION

Simple linear regression of male control, untreated and treated groups FI (preferred frequency of intercourse)(Table 80,144,Table 81,145,Table 82,146) and EP (enjoyment potential of sexual intercourse)(Table 83,147,Table 85,149Table 84,148), scores and free testosterone concentrations failed to show any significant relationships. Correlation coefficients were modest in value and for the FI scores negative.

Table 80: LINEAR REGRESSION OF MALE FI SCORES AND FREE TESTOSTERONE CONCENTRATIONS (CONTROL GROUP)

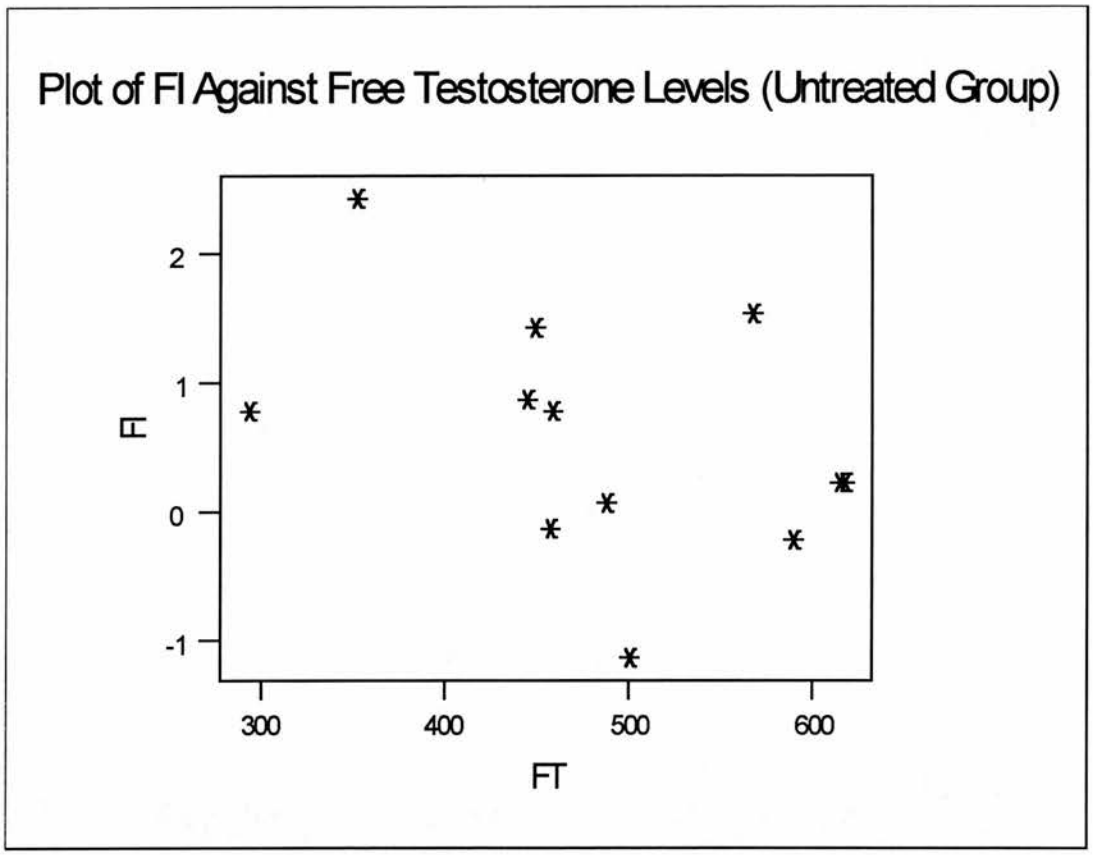


FT = Free Testosterone (pmols)

The regression equation is
FI = 2.12 - 0.00458 Free Testosterone

Correlation (Pearson) FI and FT =-0.495

Table 81: LINEAR REGRESSION OF MALE FI SCORES AND FREE TESTOSTERONE CONCENTRATIONS (UNTREATED GROUP)

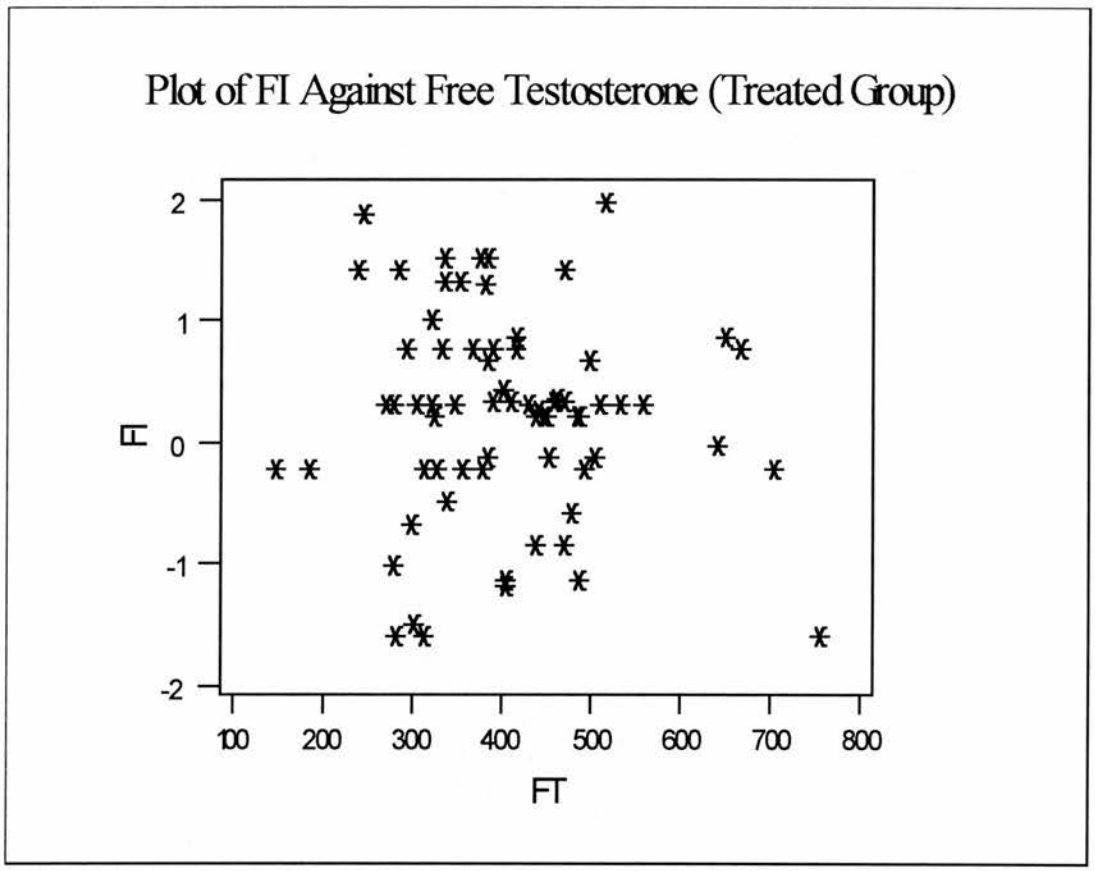


FT = Free Testosterone (pmols)

The Regression Equation is
FI = 2.31 - 0.00373 FT

Correlation of FI and FT = -0.409

Table 82: LINEAR REGRESSION OF MALE FI SCORES AND FREE TESTOSTERONE CONCENTRATIONS (TREATED GROUPS)

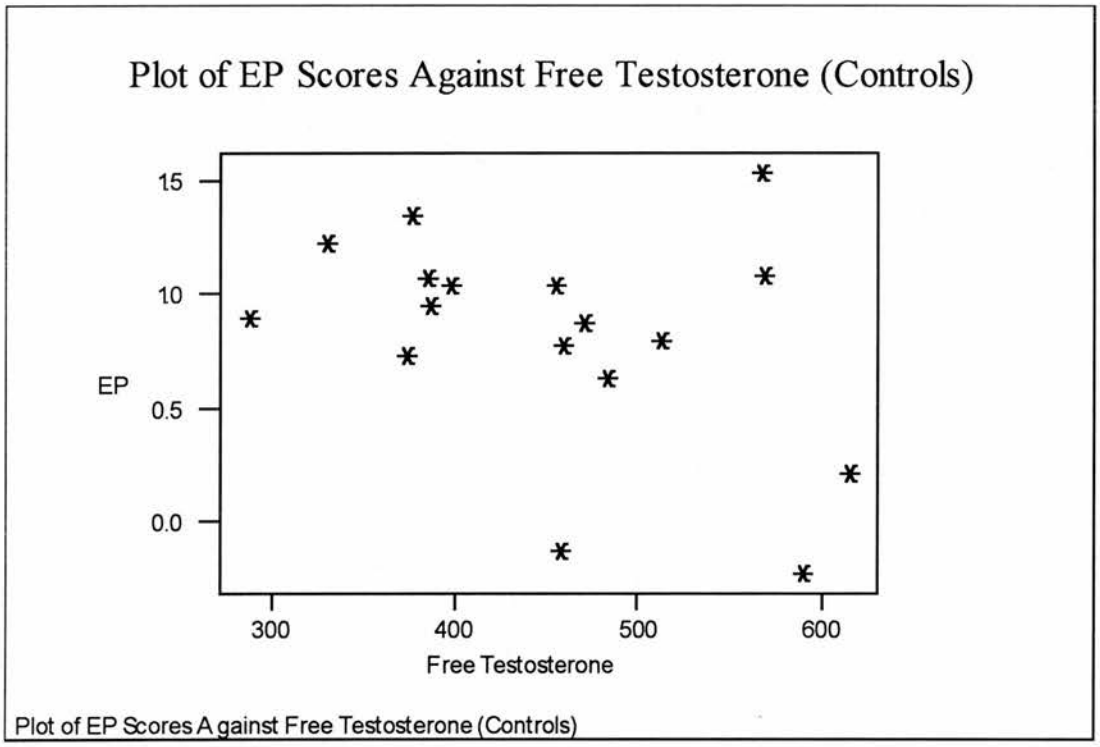


FT = Free Testosterone (pmols)

The regression equation is
FI = 0.514 -0.000605 FT

Correlation (Pearson) of FI and FT = -0.089

Table 83: LINEAR REGRESSION OF MALE EP SCORES AND FREE TESTOSTERONE CONCENTRATIONS (CONTROL GROUP)



Plot of EP Scores Against Free Testosterone (Controls)

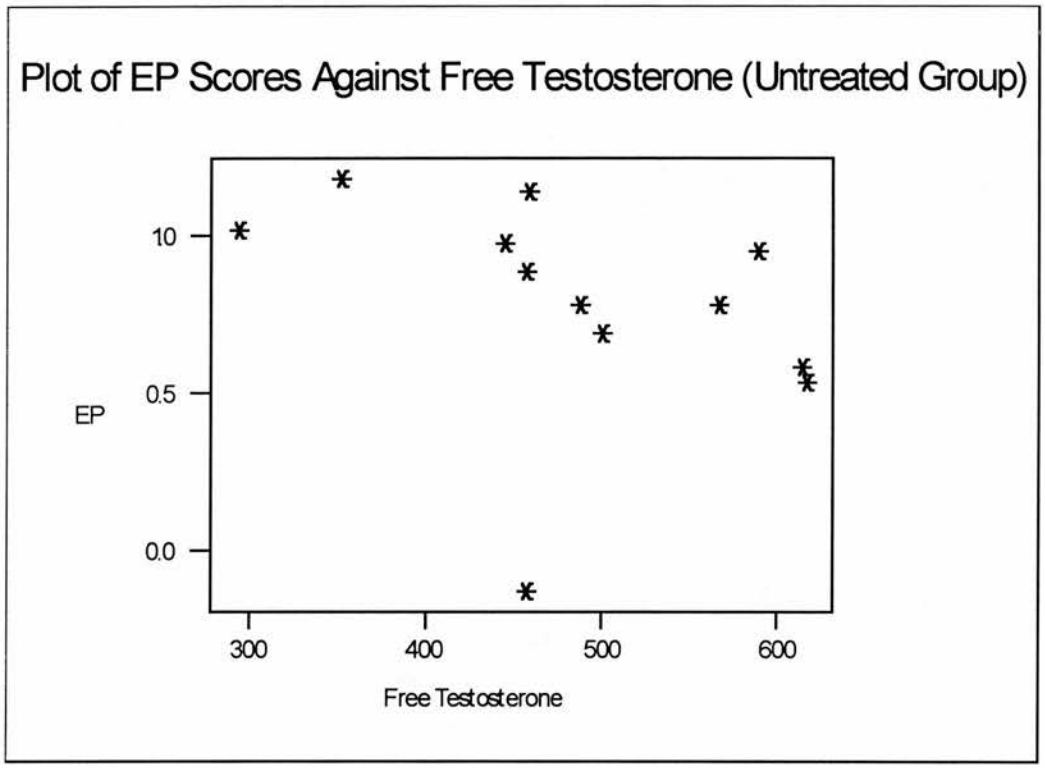
Free Testosterone in picomoles

The regression equation is

EP = 1.68 - 0.00192 Free Testosterone

Correlation (Pearson) of EP and FT = -0.256

Table 84: LINEAR REGRESSION OF MALE EP SCORES AND FREE TESTOSTERONE CONCENTRATIONS (UNTREATED GROUP)



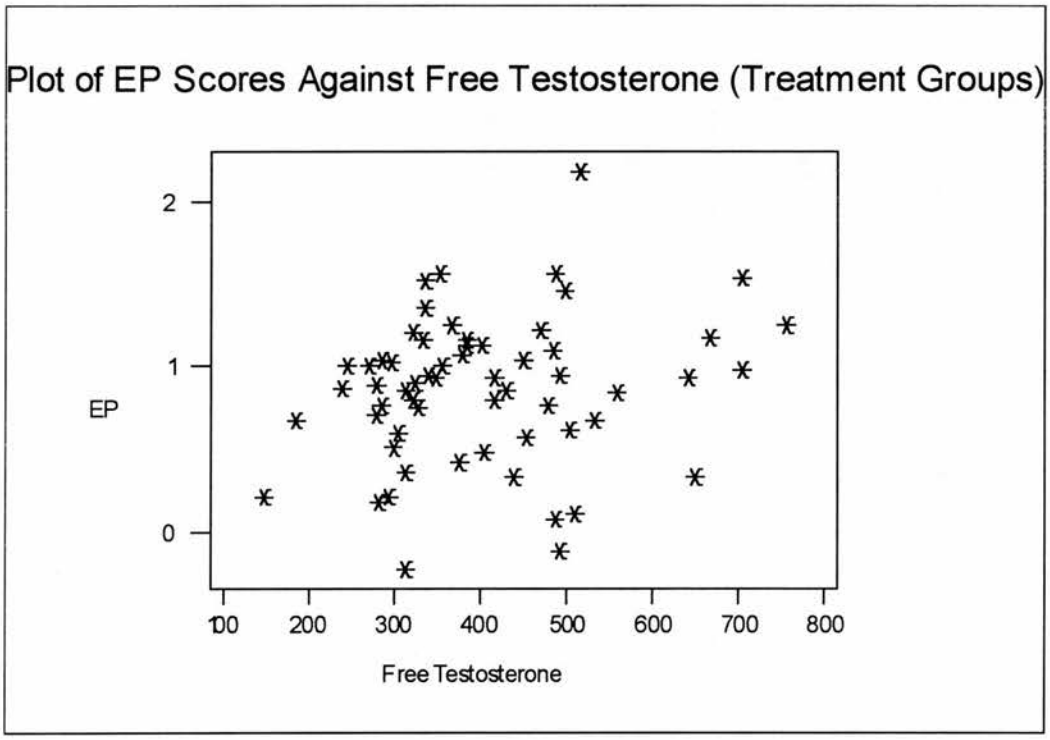
Free Testosterone in picomoles

The regression equation is

EP = 1.39 - 0.00125 Free Testosterone

Correlation (Pearson) of EP and FT = 0.743

Table 85: LINEAR REGRESSION OF MALE EP SCORES AND FREE TESTOSTERONE CONCENTRATIONS (TREATED GROUP)



Free testosterone in picomoles

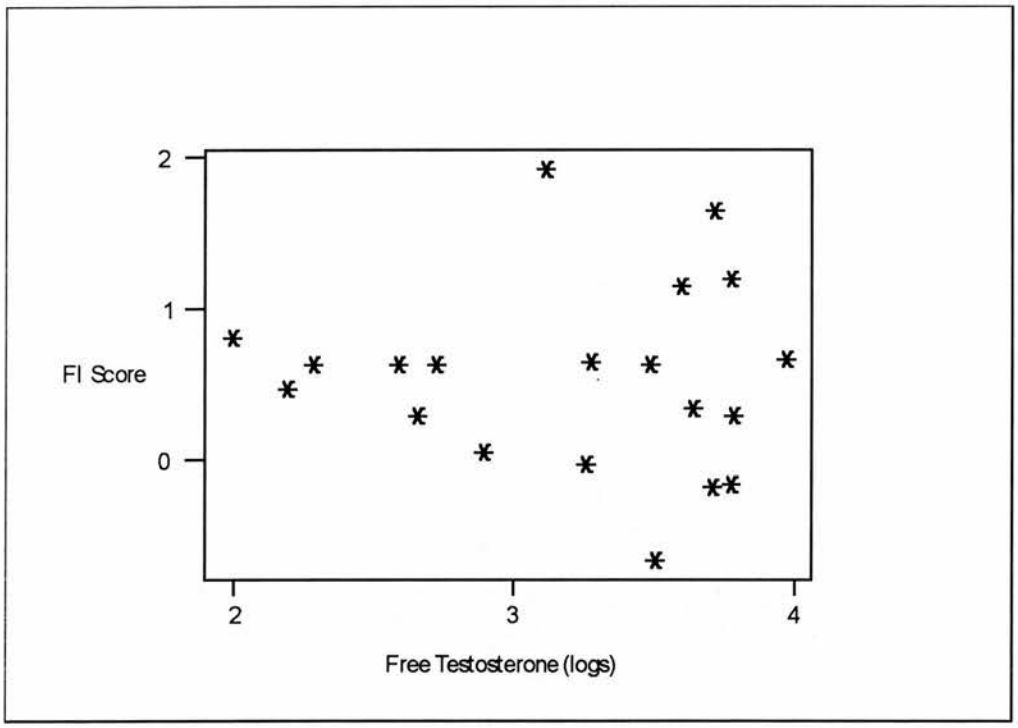
The regression equation is

EP = 0.726 +0.000425 Free Testosterone

Correlation (Pearson) of EP and FT = 0.141

Simple linear regression for the female FI (Table 86,150,Table 87,151,Table 88,152) and EP scores in the control, untreated, and treated groups (Table 89,153,Table 90,154,Table 91,155) did not show any significant relationships with free testosterone levels. Correlation coefficients were as expected small.

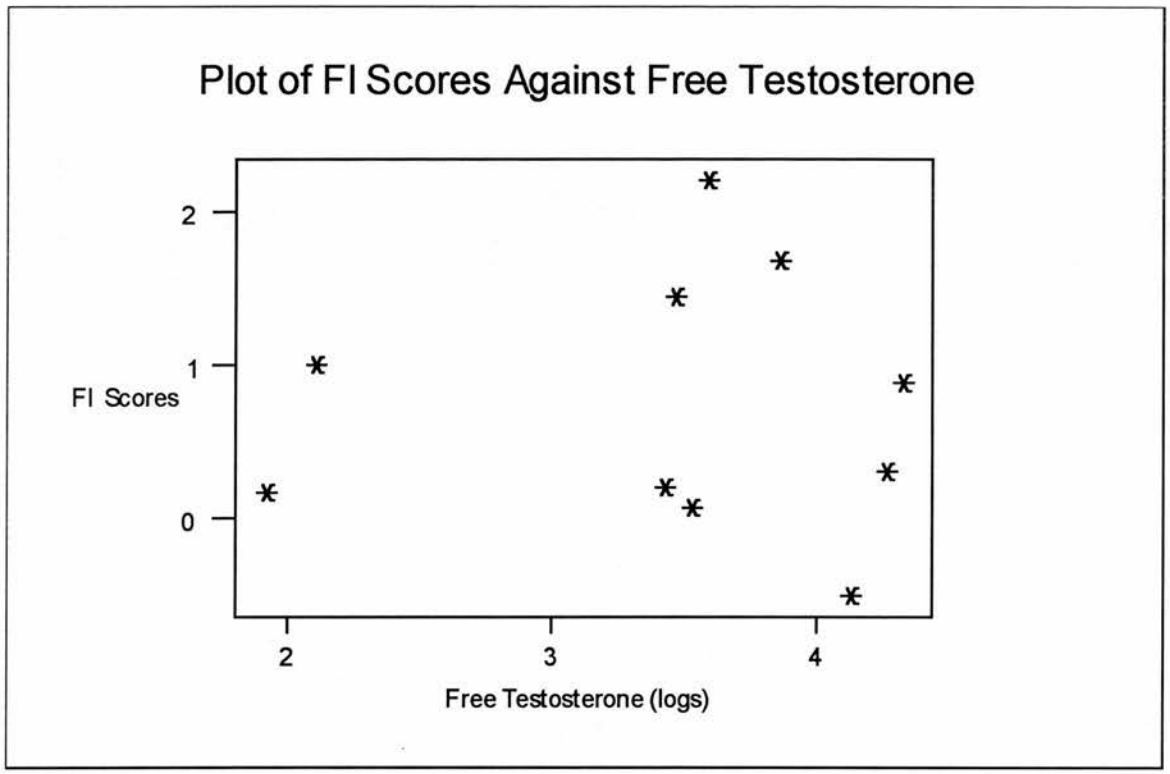
Table 86: LINEAR REGRESSION OF FEMALE FI SCORES AND CALCULATED FREE TESTOSTERONE (CONTROL GROUP)



The regression equation is
FI = 0.377 + 0.079 FT(logs)

Correlation (Pearson) of FI and Free Testosterone (Logs) = 0.08

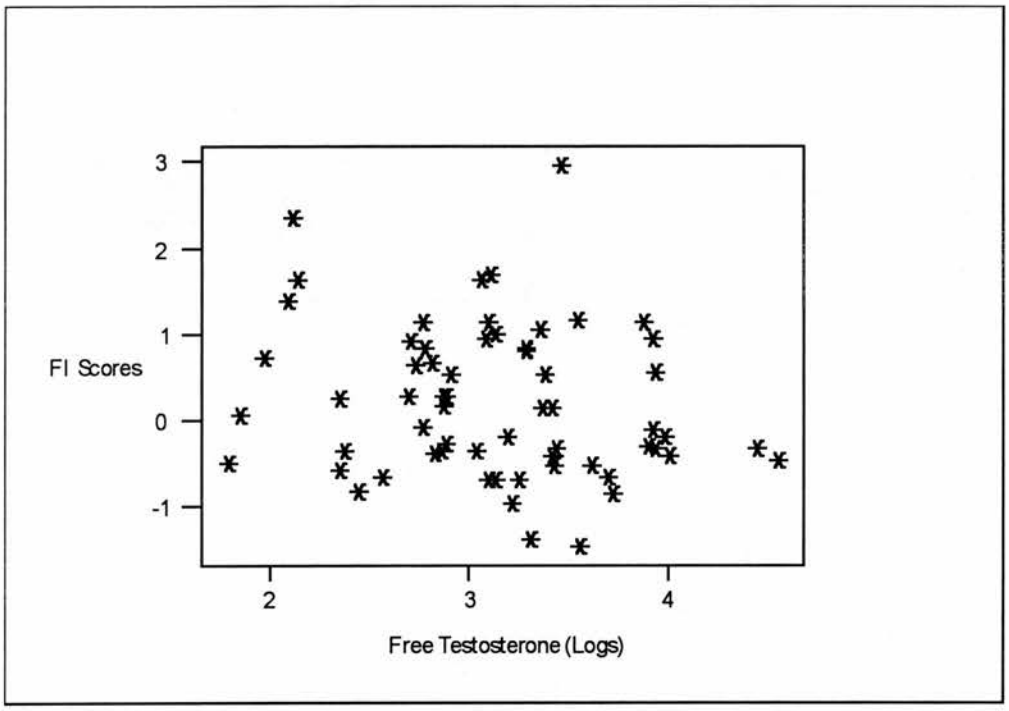
Table 87:LINEAR REGRESSION OF FEMALE FI SCORES AND CALCULATED FREE TESTOSTERONE (UNTREATED GROUP)



The regression equation is
FI = 0.74 - 0.001 FT(logs)

Correlation (Pearson) of FI and Free Testosterone (Logs) = -0.001

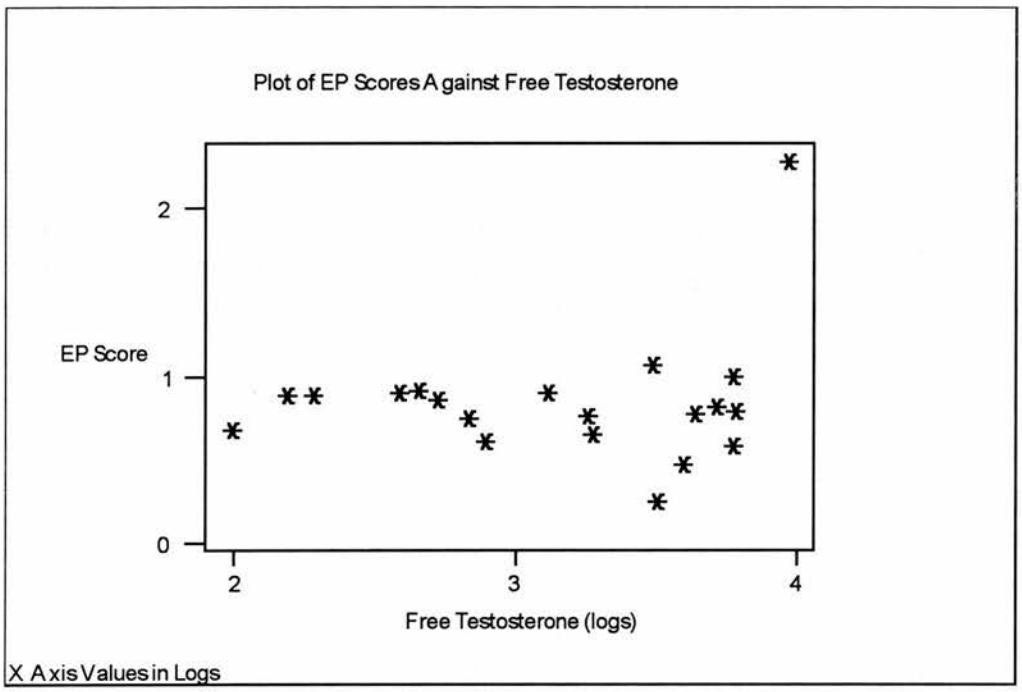
Table 88:LINEAR REGRESSION OF FEMALE FI SCORES AND CALCULATED FREE TESTOSTERONE (TREATED GROUP)



The regression equation is
FI = 1.37 - 0.375 FT(logs)

Correlation (Pearson) of FI and Free Testosterone (Logs) = -0.245

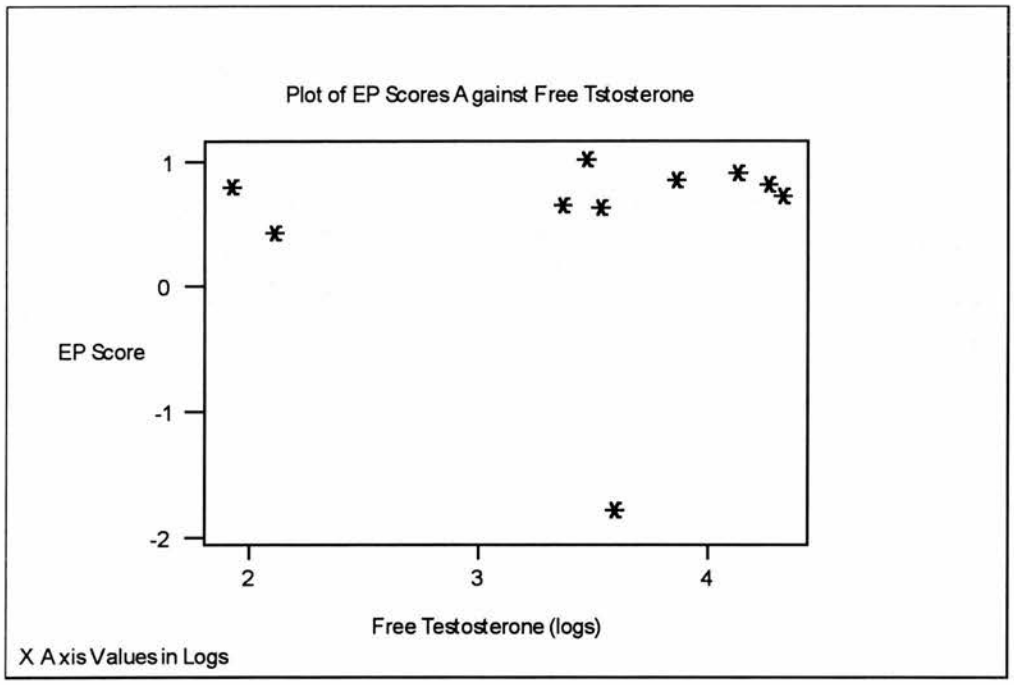
Table 89: LINEAR REGRESSION OF FEMALE EP SCORES AND CALCULATED FREE TESTOSTERONE (CONTROL GROUP)



The regression equation is
EP = 0.589 + 0.079 FT

Correlation (Pearson) of EP and Free Testosterone (Logs) = 0.187

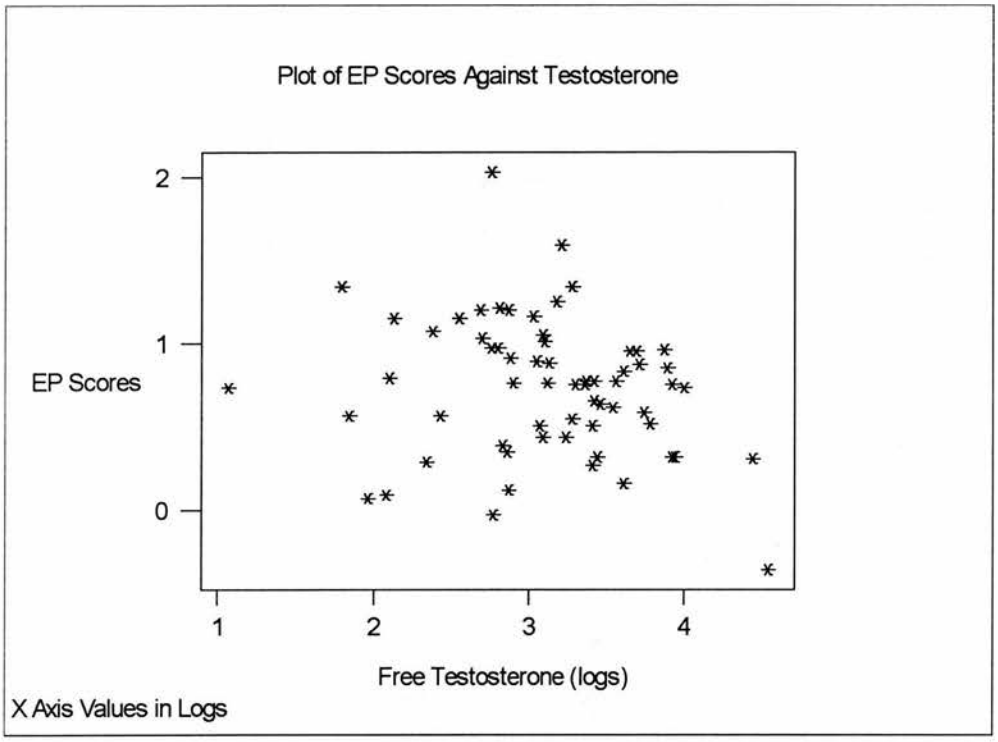
Table 90: LINEAR REGRESSION OF FEMALE EP SCORES AND CALCULATED FREE TESTOSTERONE (UNTREATED GROUP)



The regression equation is
EP = 0.38 + 0.032 FT

Correlation (Pearson) of EP and Free Testosterone (Logs) = 0.032

Table 91: LINEAR REGRESSION OF FEMALE EP SCORES AND CALCULATED FREE TESTOSTERONE (TREATED GROUP)



The regression equation is
EP = 1.22 - 0.154 FT

Correlation (Pearson) of EP and Free Testosterone (Logs) = -0.233

DISCUSSION

Most studies of sexual function in men and women with epilepsy have been undertaken in populations different from the present study. Typically the patients have been resident in or associated with institutions dedicated to the long term care of people with seizures (Gastaut et. al. 1954: Fenwick et. al. 1985). Failing that, patients have been studied as a prelude to surgical treatment of their seizures, which in this country has always been reserved for those individuals who do not respond to AEDs, and are by definition amongst the most severely affected (Falconer et. al. 1955; Blumer et. al. 1957; Taylor 1969; Taylor 1971). Patients in this study, by contrast,were living independently in the community.

Statistical examination of both SES 1 and 2 and their attendant subscales showed that men and women with epilepsy, especially those receiving AED therapy were more accepting of a strict moral code compared with the controls and less open to psychosexual stimulation. A similar pattern has been observed by other workers (Morrell et.al.1994) who using the Sexual Behaviour Inventory and Beck Depression Inventory found both men and women with seizures had fewer sexual experiences, and that women, but not the men reported various imagined activities as significantly less arousing and more anxiety provoking compared with healthy controls. In this study the only behavioural assessment made of the subjects was the SES scores. In retrospect an attempt at appraisal of other psychosocial factors could have been made. A questionnaire pertaining to mood such as the Multiple Affect Adjective Check List (MAACL) (Zuckerman et. al. 1983) would have been useful, as indeed would questions aimed at exploring the individuals attitudes to their own epilepsy. Both mood and the person's feelings about their condition at the time the SES questionnaire was completed

could have affected the scores attained. Confidence intervals in the female untreated group tended to be wide with much less variation in the treated group with little overlap between the treated and control group. There was overlap between the untreated and treated groups on the SES 2 unweighted scale, and on the EE scale of SES 2. A similar phenomenon was seen in the male results with a greater degree of overlap in the SES 2 scores so the differences are not quite as convincing statistically as those recorded for the women (see statistics index)

One obvious explanation of the differences observed on SES 1 and 2 scores is the differences between the male and female control groups in their rates of further education compared with the untreated and treated groups. In addition the men in the treated group were significantly older than the control and untreated men and the women in the untreated group were significantly younger than both the controls and treated women, both of these parameters have an effect on SES scores (Frenken and Vennix 1981). In this type of study the composition of the control group is crucial, yet rarely is perfection achieved. Personal details of the male and female control groups can be found in appendix one, in which it can be seen that our controls interpreted the term "further education" in a variety of ways. One man claimed, correctly in his opinion- that he had had further education on the strength of a recently completed day release course at the University of Paisley. The same held true of the women who had trained as hairdressers, nurses and bank employees, most of whom had attended day release courses, yet had never left home or the environment in which they had grown up.

The mode of recruitment of the controls may also be criticised. Other studies e.g. Ndegwa et.al.1986 and Jensen et.al. 1990 have used consecutive patients attending general practitioners as controls. This method assumes that the control individual is not presenting to the GP with physical ailments which are being used to mask psychological or psychosexual problems. In this study the closest GP practise was located in a prosperous part of Glasgow where a large proportion of the patients were in high paid employment. In addition, few local practises could have accommodated the two personal computers, blood tubes and centrifuge necessary during the time of the study. Thus working on the assumption that the waiting areas of large teaching hospitals are as demographically heterogeneous as a typical GP's waiting room the author opted for the technique described in the methods section.

One further caveat should be added about the make up of the controls in that it was assumed that all respondents were heterosexual. It was not deemed appropriate by the leader of the research team to ask questions about individuals sexual orientation. To circumvent this during the period of instruction on the personal computers the respondents were told on at least one occasion that the questionnaire was designed for heterosexuals.

We were unable to procure a set of normative values for the SES scales obtained on a Scottish population. Frenken and Vennix devised their scale in 1981 on what they described as a " broad middle class " in the Netherlands. This lack of a set of Scottish

normative values has, however, not precluded the SES from being used by other workers in this field (Bancroft et.al. 1991), but some caution is required when considering the results obtained.

Multiple regression for male SES 1 and 2 unweighted scores showed that as Frenken and Vennix predicted further education had a significant predictive value, age, however did not, the same pattern was seen on two of the SES 1 subscales, the MS and PA and the AE subscale of SES 2. Although the female control group had a higher proportion of women who had received further education compared with the men multiple regression did not show any significant predictive effect of this parameter on unweighted SES 1or SES 2 scores, unlike the men, although it did exert a significant influence on the MS and SS subscale scores of SES 1 and the IS subscale of SES 2. Thus these results must be interpreted in the light of these differences between the study and control groups.

Putting aside the disparities between the control and study groups there are other reasons why individuals with epilepsy might embrace a strict moral code. One is as a way of avoiding sexual experimentation whether due to genuine lack of libido or fear of the effects it might have on their seizures. Taylor (1969) described several cases of individuals with CPS avoiding marriage and by implication sexual intercourse - some of the cases dated from the early sixties - by dint of elaborate excuses, such as one man who was almost constantly engaged, but contrived to break off the relationship

whenever it looked like coming to fruition by accusing each woman in turn of being too financially demanding.

The roots of this eschewing of sexuality may be laid early on and inadvertently by the parents of the child with epilepsy. Gagnon et. al.(1973) proposed the concept of a psychosexual script, written for us at an early stage and reinforced by our parents such that we learn from them what is considered appropriate behaviour at certain stages of our development and dictates the sex and kind of person we will find attractive on gaining full maturity. If this process is subverted then full maturity is not achieved, and assumes that the hypothetical child is healthy. There is ample evidence that children with epilepsy exhibit more neurotic traits than their siblings (Stores et.al. 1978; Matthews et.al. 1982; Hoare et.al. 1991) and require more affection and reassurance. Matthews 1982 study also reported that children with seizures considered themselves less popular than their contemporaries, a belief which if carried into adolescence could profoundly influence social interaction with the opposite sex. Another study (Long 1979) that found parents of children with epilepsy anticipated the child would have more emotional problems and less choice of a job on reaching adulthood. Thus a child with epilepsy may have a "script" written for them which implicitly precludes sexual activity or the formation of long-term intimate relationships.

The observation that for the men there was no statistically significant difference in mean scores on SES 3 (Sexual Motivation Scale) and its subscales is interesting as it implies that the differences in SES 1 (Sexual Morality Scale) and 2 (Openness to Psychosexual

Stimulation Scale) between the groups not withstanding those men that do establish relationships appear no different in their preferred frequency of intercourse, enjoyment of it or orgasmic adequacy. This might imply that there are two populations of men, as Bergen et.al.(1992) suggested there were of women, one of whom had normal libido, and one who appeared sexually indifferent, and that those men with normal sexual interest were able to establish relationships in the same way as their healthy peers. Against that, however, is the observation that marital status had no predictive value on multiple regression of SES 1 and 2 and their subscales. The women presented a slightly different picture with the treated group attaining a significantly higher mean SES 3 score than both the controls and untreated groups. The women in the treated group rated their orgasmic adequacy as significantly less than that of the control group. Frenken and Vennix (1981) point out that orgasm is dependant on the individual and inner experiences and are not dependant on interaction with the partner in the way the EP subscale is. This difference between male and female results may be the result of a specific physiological deficit sustained by women with seizures or it may reflect the findings of Morrell et.al.(1994) that women with epilepsy are more likely to find certain aspects of sexual interaction anxiety provoking. Before concluding this, however, attention has to be drawn to the wide confidence intervals (see statistics appendix) obtained in this analysis and treat the apparent difference in OE scores with some caution.

The results of the SES 4 subscales - the attraction to marriage scale - showed that the men in the treated group appeared to enjoy a greater marital satisfaction than the other

two groups. Overall the controls seemed as attracted to their marriages' than the other groups. This a little surprising at first glance given that men in the treated and untreated groups tended to espouse a stricter sexual morali and be less open to psychosexual stimulation, both traits that might cement a relationship. Men in the treated group were older than in the other two groups and which might be expected to lend some extra stability to the marriage. Against that it the finding that age at time of study had no significant predictive effect on the MS and ES scores. A note of caution must be added about the attraction to marriage scores in that the confidence intervals were wide with some overlap. The women of the treated group appeared even less attracted to their marriage than their male counterparts, age cannot be so easily invoked given that the female controls and treated group had mean ages of within a year of one another. The treated women rated their partners to be less satisfactory than both controls and untreated, this may reflect the anxieties caused by sexual activity that Morrell et.al. (1994) described or it may be a manifestation of their partners fears about making "demands" on a wife he considers an invalid.

As with men the confidence intervals for the AE and ES subscales are wide, and some caution in interpreting them must be made.

The sedative and cognitive effects of AEDs have attracted much attention over the years (Trimble 1987), with evidence that CBZ and PHT can produce reductions in psychomotor performance and memory respectively (Gillham et. al. 1988; 1990) particularly when used in combination with other AEDs. More recent work suggests

that patients taking CBZ or PHT as monotherapy sustain only marginal impairment (Pulliainen et. al. 1995). Saunders and Rawson (1970) found one case of reduced libido which they felt attributable to drug therapy. Fenwick (1985) found very little relationship in his study, but reported that CBZ was associated with sexual dreams. Some of the literature describing hyposexuality (Gastaut et. al. 1954) and others was written before the introduction of CBZ or VPA for epilepsy, and in Demerdash's study the majority of women were being treated with a barbiturate or PHT. Given that there seems to be some degree of impairment of psychomotor responses on laboratory testing especially on visual tracking in the case of PHT (Gillham et.al.1990) one might have expected to see some effect of AED levels on AE subscale scores. No significant effects were demonstrable and highlights the fact that what goes on in the psychology laboratory may not have a great deal of relevance to the individuals everyday functioning. Their findings on PHT notwithstanding Gillham et.al.(1990) and latter Pulliainen et.al.(1995) both make the point patients on monotherapy who are tolerating their drug regime and are not clinically toxic exhibit only slight deficits of memory and psychomotor function. Thus our observations are not entirely surprising. A better design for this part of the study might have been to administer the SES to those newly diagnosed men and women before the inception of therapy and then to readminister it several months later in order to ascertain whether there were any effects on arousal on exposure to erotic material.

Previous studies have tended to report reduced sexual interest in those individuals suffering form CPS (Gastaut et.al.1954; Blumer et al 1967; Taylor 1969;

Saunders et.al. 1970; Cogen et.al.1979; Jensen et. al. 1979; Shukla et.al.1979; Pritchard 1980; Demerdash 1991). The author found very few differences on any of the four SES subscale scores, for both men and women, certainly none which pointed to individuals with CPS being less open to arousal by erotic imagery or any more or less open to psychosexual stimulation. Those men and women with CPS did not differ in their preferred frequency of intercourse or enjoyment of it. This is not the first time such an observation has been reported. Toone et. al. in 1984 whilst finding diminished sexual interest in two groups of men with epilepsy one living the community and the other in an institution reported that men with CPS were no more likely to be hyposexual than men suffering from other forms of seizure. A major problem in this field of study is the correct diagnosis, not only of epilepsy, but of the type of seizure from which the patient is suffering. At the time of recruitment all of the patients were asked to describe their seizures, and wherever possible an eye witness account was obtained either from an accompanying relative or friend or from the case notes. It is still perfectly possible, however, that some of the patients were incorrectly assigned. This is particularly so with the undefined group where there is the possibility that individuals with NES were recruited. This is a risk all workers in the field of epilepsy face, however, no matter how strenuous the efforts of researchers to exclude such patients. It is also possible that people labelled as primary generalised epilepsy on the grounds of history and EEG may have had partial onset of seizure with such rapid generalisation that they had no memory of the aura. The author is satisfied, however, that the majority of our patients had their seizure type correctly classified.

Seizure frequency might be expected to affect the individuals desire for intercourse given the attendant social embarrassment and, in the case of tonic-clonic seizures, physical sequelae of seizures yet the male group showed no significant relationship between the number of seizures in the preceding three months and FI scores. The situation with the women was slightly different in that there was a significant P number but the correlation coefficient and R^2 were modest. These observations are perhaps a little surprising given that an improvement in sexual function after temporal lobectomy for CPS has been reported (Blumer et.al.1967). Blumer's patients were, however, by definition more severely affected than study patients which may in part explain the difference. Blumer also states that his patients experienced an improvement in social function which would improve individuals chances of finding a partner. Seizure frequency is not as easy to assess in people living in the community. The investigator depends on the goodwill and honesty of the patients in keeping accurate seizure diaries. Epilepsy by its nature relapses and remits, and three months is a short time over which to assess seizure frequency given that people with CPS often exhibit clustering of seizures. In addition those patients living alone may sustain seizures of which they are unaware. Yet asking patients to keep accurate seizure diaries for longer periods runs the risk of the patient forgetting to fill them in or worse completing them in retrospect to keep the doctor happy.

Tonic-clonic and some complex partial seizures are known to cause derangement in prolactin, thyrotropin, cortisol, growth hormone, and vasopressin levels (Pritchard et al 1983; Aminoff et al 1984; Dana-Haeri et al 1983; Rao et al 1989).

These changes, however, are short-lived and unlikely to make a significant difference to sexual interest or reproductive function, neither is there any evidence in the literature that the changes in hormone levels observed exert a cumulative effect although that is a possibility.

Given there is evidence that cognitive impairment occurs in people who suffer a considerable number of attacks (Dodril et.al.1984) one might expect to find those individuals sustaining frequent seizures would be less open to arousal by erotic scenes in the visual and print media, yet we found no evidence of a relationship between AE scores and number of seizures in either sex. The author did not ask subjects about sexual activity during times of maximal seizure activity as the aim was to gain an overall impression of the individuals sexuality, but it would seem reasonable to assume that it would diminish at those times. The results of FI and AE scores in relation to seizure numbers could be seen as indicating that any diminution in libido observed in people with epilepsy is due to the underlying structural or neurochemical abnormality causing seizures rather than the attacks themselves.

Lindsay et. al. (1979) reported on a cohort of children all of whom had developed seizures in childhood, and had been referred for specialist assessment usually for behavioural problems. Of the 66 children followed up only 41% of the boys who had developed epilepsy before puberty were married, as opposed to 92 % of the women. Those boys whose seizures had remitted before puberty were most likely to marry. The unmarried men in this group, all of whom had limited sexual activity or were frankly

indifferent, had not, with one exception remitted until after puberty. Similar observations were made by Gastaut (Gastaut et. al. 1954; Fenwick et. al. 1985). One obvious suggestion for this observation would be that the earlier the child experiences the psychosocial problems associated with seizures the less likely he is to separate emotionally from his parents and develop fully adult relationships. Although if that were the whole story it would be difficult to explain the marked discrepancy between the men and women. What role seizure activity in the amygdalar-hippocampo-hypothalamic axis plays is a moot point. As discussed in the introduction this area of the brain plays a vital part in non-human primate social behaviour and neuroendocrine control, and must be intact for social and sexual intercourse to be normal. That early onset does not seem to exert such a deleterious effect on women may point to an element of sexual dimorphism. Taylor (1969) investigated the differences in incidence of mesial temporal sclerosis (MTS) presumed due to febrile convulsion, between the sexes, and found the period of greatest risk was in the first two years for girls and four years for boys. Thus boys are at greater risk of MTS and this may explain the differences in incidence of marriage between the sexes. One must bear in mind that the three studies cited all involved men who had been living in institutions or who had been referred for specialist help because of intractable seizures or behavioural problems. Of the 66 men and women Lindsay and colleagues reported on 34 had suffered either severe head injury or meningitis before the onset of CPS, or had had a complicated febrile fit. We made no inquiry into the aetiology of our patients seizure, such a line of inquiry might reveal differences in rates of marriage and SES scales. Both Taylor (1969) and Morrell (Morrell et.al. 1994) allude to the fact that there appears to be a deficit in arousal in

people with epilepsy. Morrell (1994) demonstrated that in men and women with epilepsy genital blood flow increased when they were exposed to erotic films but to a significantly lesser degree than in controls. We did not study the physiological responses of our patients, but linear regression of AE (Arousal by Erotic Imagery) scores against age at onset of epilepsy failed to show any significant relationship for either men or women. We did not, however, analyse the CPS group separately, which might have given a different result. There were differences, however, when the rates of marriage were examined with men who had developed seizures in the first two decades being proportionately less likely to be married than those men who sustained their first seizure after the age of 21. The picture is less clear in the women. There is a trend for those women who developed epilepsy before the age of 21 to be less likely to be married than those who acquired the condition thereafter. One reason for this observation may be that the responsibility for taking the initiative still in the main lies with the male, thus any lack of sexual interest may be more likely to be reflected in male rates of marriage. Rates of marriage are a crude way of examining sexual interest, however, if an individual is married it does imply that at some point they exhibited some form of sexual awareness.

Raised SHBG in association with AED therapy has been previously reported (Victor et. al. 1977; Barragry et. al.1978; Toone et. al. 1980; Connell et. al. 1984; Herzog et. al. 1986; Isojarvi et. al. 1988; McPhee et. al. 1988; Isojarvi et. al. 1991). The reasons for this are unclear. Enzyme inducing drugs such as CBZ, PHT, and rifampicin are known to increase SHBG by stimulating liver production (Brodie et. al. 1981)

which may produce an alteration in TT/FT ratios with a consequent fall in FT leading to a further rise in SHBG synthesis (Anderson 1974). An alternative explanation is that enzyme induction leads to increase in metabolism of FT which in turn stimulates SHBG production (Anderson 1974; Toone 1976). Thus the male SHBG results are in line with previous work in this area. It is interesting to note that VPA, which is not an enzyme inducer (Gram 1991), had a significantly lower mean SHBG than both PHT and the polytherapy group. Dowsett et.al. (1985) reported that levels of SHBG were higher in the luteal phase of the menstrual cycle compared with the proliferative. This was certainly the case in the untreated women although the difference did not reach significance. Interestingly there was not such a clear cut difference between proliferative mid-cycle and luteal phases in the treated group. As with the men those women receiving AEDs had significantly higher SHBG than those who were not. Because of the design of the female study no comment can be made about the effect of individual AEDs on SHBG, but as the first report of raised SHBG in association with AED therapy was made in women (Victor et. al. 1982) it is safe to assume that the difference is drug induced. Some caution must be extended in examining the female hormonal data. Ideally the author should have sampled each woman on the same day of the menstrual cycle. This was not possible as subjects were attending a routine clinic, in addition the author did not measure progesterone levels to ascertain whether the women were ovulating or not a factor which would influence our results. Secondly the author did not measure the women's' weight or ask about their smoking habits both of which can influence sex steroid levels.

Raised TT in association with AED therapy has been noted on several previous occasions (Barragry et. al. 1978; Toone et. al. 1980; Toone et. al. 1983; Toone 1986; Daniels et. al. 1984; Herzog et. al. 1986; Isojarvi et. al. 1991), and is presumed due to raised SHBG levels. Rifampicin when administered to healthy male volunteers induces a rise in both SHBG and TT (Brodie et. al. 1981). The female treated group had a significantly higher TT than the untreated subjects, and the male VPA group had a significantly lower mean TT than the PHT and polytherapy groups. There was no significant difference between the CBZ and VPA groups which given that the former is an enzyme inducer is somewhat surprising. CBZ when given to healthy volunteers causes rise in SHBG, but a fall in TT and FAI levels which return to normal after the third week of therapy (Connell et.al. 1984), it also induces the hepatic microsomal mixed function system (Rapeport et. al. 1983), the same system that metabolises testosterone (Vermeulin 1979) which may explain the fact that TT in this group is not as raised as the PHT and polytherapy groups. PHT is, however, also a hepatic enzyme inducer, yet, mean TT for that group is significantly higher than that of the CBZ group. One study (Luhdorf et.al.1977) showed a rise in TT in patients treated with VPA. So invoking the effects of AEDs on the hepatic microsomal enzyme system may not be the whole explanation for these results.

As FT is considered to be the physiologically active moiety of circulating testosterone the effects on it of AEDs are perhaps of greatest interest given the implications for sexual function. The observation that both men and women taking AEDs had calculated FT levels within the normal range quoted by the local endocrine laboratory makes it

unlikely that any deficit in sexual interest can be largely blamed on AED drugs. Gooren (1987) showed that levels TT ranging from 5 nmol/l to 11 nmol/l or above were required to maintain sexual interest in hypogonadal men, and Salamimies (1982) reported that TT levels 6 nmol/l to 12 nmol/l were required. The lowest TT recorded in the male group was 7 nmol/l. Taking 6 nmol/l as the "androgen threshold " for men, and assuming that 1% to 3% of it is FT then the threshold would lie between 120 pmol/l and 360 pmol/l, again above our lowest calculated FT result.

There are two possible ways in which the calculated FT might be misleading. The first lies in the equation we used. Although it has a high degree of correlation with measured FT it is possible that some of the values fell below the lower end of the physiological norm, the second and less likely is that seizure activity in some way alters androgen sensitivity either peripherally or centrally and a higher degree of FT is required to maintain the same degree of sexual interest. This study is not the first to report normal FT levels in individuals taking AEDs (Isojarvi et. al. 1988 and 1991) and this observation calls into question the value of FAI in this field given that FT is accepted as the physiologically active component (Anderson 1974). FAI calculations use SHBG as the denominator, given that certain AEDs cause a rise in SHBG concentrations it is not surprising that FAI is noted to drop in individuals prescribed them. Reduced FT is said to cause a rise in SHBG (Anderson 1974) which stimulates further SHBG production which in turn binds more FT causing a fall in its concentration thus promoting more SHBG production. Neither in this study or in others (Isojarvi et al 1988 and 1991) has this been found to be the case. Either the concept of reduced FT inducing SHBG is too simple or the men in these studies maintained their FT through other means.

Competition between AED and testosterone for binding sites on albumen, which has a large capacity but low affinity for testosterone (Anderson 1974) is one possible mechanism.

Another observation of interest was that the treated women had a significant rise in midcycle calculated FT compared with the proliferative and luteal phases. This has been noted before (Goebelsmann et.al.1974) and has been suggested as one of the underlying mechanisms for the increase in sexual activity reported at the time of ovulation (Adams et.al. 1978). Given that SHBG begins to rise at about this time (Dowsett et.al. 1985) and that that rise might be exaggerated due to AED therapy a reduced midcycle FT might have been anticipated, against that, however, is the calculation by Dowsett et.al.(1985) that any change in FT would be small (see introduction).

In the male group the author found no evidence of a significant relationship between FI and EP scores and calculated FT. This is not surprising given the work of Gooren (1987) and Salamimies (1982) showing that above a critical level FT concentration was irrelevant in men. Separate analysis of the control, untreated and treated group showed that men with epilepsy whether receiving AEDs or not behaved in the same way as the controls which would tend to go against the hypothesis that seizure activity might in some way reset androgen receptors. These results should be treated with some caution as the numbers in the control and untreated groups are small.

As was explored in the introduction the relationship between FT, sexual motivation and actual intercourse in women appears to be more complex than in men, with the effects of FT on these factors being over shadowed by psychosocial influences. There is evidence that FT is positively associated with frequency of intercourse (Bancroft et.al.1991) in women taking the pill but not in those who are not. As none of the women whose hormonal status we examined were taking the OCP our finding that there were no significant relationships between FI scores and calculated FT is in line with previous work. Given that the link between androgen status and sexual interest is more easily influenced by extraneous factors in women it is important that some inquiry into the respondents overall psychological status is made. In retrospect an additional questionnaire such as the multiple affect adjective checklist (MAACL) (Zuckerman et. al. 1983) should perhaps have been included to explore this. Neither did the author include OCP status as one of the independent variables in our multiple regression of the SES scores. As a negative correlation between FT and restrictive morality has been reported in women taking the OCP (Bancroft et.al.1991) some significant effect might have been anticipated. Because women report different levels of sexual interest at different phases of the menstrual cycle (Bancroft 1989, page 118) it would have been useful to correlate FT levels for women in the proliferative, ovulatory, and luteal phases with their respective FI and EP scores. This was not possible, however, because of the small numbers involved, given that only those people with long-standing partners were asked to answer these two questionnaires.

Dehydroepiandosterone sulphate (DHEAS) is of adrenal origin (Ducharme et. al. 1982) and is a weak androgen compared with testosterone and 5 α dihydrotestosterone (Johnson et. al. 1991). Our findings that men taking CBZ, PHT or polytherapy had significantly lower mean DHEAS levels than the controls, untreated and VPA groups has been previously reported (Connell et. al. 1984; Levesque et. al. 1986). Although no comment can be made about the effects of individual AEDs on female DHEAS levels those women in the treatment group had significantly reduced levels in comparison with the untreated females. The same mechanisms invoked to explain the changes in TT and FT observed in people taking these drugs have been advanced to explain the changes in DHEAS. Namely increased liver metabolism due to enzyme induction or increased SHBG synthesis. This would seem reasonable given the finding in this study that the VPA monotherapy group had significantly higher DHEAS levels than the PHT, CBZ, and Polytherapy groups. An alternative explanation for the reduction in DHEAS observed in this, and other studies may be that there is a direct toxic effect of PHT and CBZ on the adrenal glands. There was a significant difference in mean DHEAS concentration of the polytherapy group compared with the CBZ group which might imply that PHT alone and combinations of AEDs are more toxic to the adrenals another possibility is that people on PHT and polytherapy may have had the disease longer than those on VPA and CBZ.

As AND is converted to testosterone in both sexes it is not surprising that there was no significant diminution in levels in association with AED therapy in either sex. Indeed there was an ovulatory phase rise in levels in both female groups, reflecting the fact that

50% of AND comes from the ovary whilst the remaining 50% is derived from the adrenal (Baird et.al.1974). This observation of preserved AND concentrations in the face of AED therapy is an important one in women where AND is the most abundant androgen (Bancroft 1989, page 27). It also suggests that adrenal toxicity cannot be the sole mechanism for the reduction in DHEAS seen in individuals of both sexes taking enzyme inducing AEDs. In the men the finding of a significantly higher mean AND concentration in the PHT group in comparison with the control, untreated, VPA and CBZ groups is an interesting one. Herzog and colleagues (1991) noted that men treated with PHT had significantly greater estradiol levels than both a healthy control group and a group of men with untreated seizures. They postulated that PHT induced aromatase thus increasing the conversion of testosterone to estradiol. AND is, however, converted by aromatase to estrone then by 17 β hydroxy steroid dehydrogenase to estradiol (Johnson et.al.1991, page 37). If PHT induces aromatase then a drop in AND levels might be expected. Another possible mechanism for this observation of raised estradiol levels in men treated with PHT lies in the structure of the hydrantoin ring which is similar to the imidazole ring of cimetidine. Cimetidine is known to inhibit the P 45 cytochrome oxygenase system resulting in raised estradiol levels (Galbraith 1989), by virtue of its imidazole ring if this were to lead to an accumulation of estradiol then 17 β hydroxy steroid dehydrogenase might be inhibited by dint of simple enzyme kinetics leading in turn to a build up of estrone and and inhibition of aromatase.

Overall it would appear that enzyme inducing AEDs are responsible for changes in SHBG,TT and DHEAS levels in people treated with them. The author found no

evidence that FT levels in either sex were compromised, and no evidence that these calculated FT values correlated with preferred frequency of intercourse and enjoyment potential of sexual interaction.

SUMMARY AND CONCLUSIONS

At the outset of this study we defined certain objectives based on evidence from other groups suggesting that individuals with CPS have a deficit in arousal which in turn leads to a diminution in sexual interest and activity. It is impossible to examine every aspect of the individual's sexuality, but certain facets were open to scrutiny. A persons sex life can be circumscribed by social circumstance and the attitudes learned from parents and peers.

Women receiving AED therapy had higher unweighted SES 1 (Sexual Morality) scores than both the control and untreated groups, with analysis of the subscales of SES 1 also revealing a tendency for those women in the treated group to espouse a stricter sexual morality. Multiple regression showed further education and religious affiliation had significant predictive effects on these scores. A similar pattern was seen when the female SES 2 (Psychosexual Stimulation) unweighted and subscale scores were examined with those women taking AEDs appearing to find exposure to erotic imagery less pleasing and reporting lower levels of interpersonal sexual attraction compared with controls. Once again further education and religious affiliation appeared to be predictive in some of the responses given by the women to these questions. Analysis of the subscales of SES 1 and SES 2 showed women in both the CPS and PGE groups espoused a stricter sexual morality within marriage with a tendency for the women suffering from CPS to be more rigid in their adherence to morals than other seizure groups and controls. These observations were not, however, reflected in the unweighted SES 3 (Sexual Motivation) and SES 4 (Attraction to Marriage) scores where women taking AEDs expressed a significantly greater sexual motivation in relation to their

partner than both the controls and untreated groups. With the exception of the OE (Orgasm adequacy during intercourse and its evaluation) subscale of SES 3 there were no significant differences between the groups indeed the treated group achieve a lower mean FI (preferred frequency of intercourse) score than the controls implying that wished to engage in sexual activity more frequently than those in the control group, interestly the treated groups mean EP (enjoyment potential of sexual interaction) score was higher than both the controls and untreated suggesting that they did not derive the same pleasure from sexual activity as those other groups, an observation that may be explained by the difference in OE scores. Overall it appeared treated women reported as strong if not slightly stronger sexual motivation as controls. There were no significant differences between the seizure type groups and controls on any of the SES 3 subscales, nor were there any clear trends. The mean scores of all three study groups fell within the range designated as " moderate attraction to marriage ", examination of the subscales showed that women in the treated group had significantly lower mean AE (attitude to extramarital involvement) and ES (evaluation of partner as sexual partner) scores than both controls and untreated in the case of the AE subscale and compared with the untreated for the ES subscale implying that the treated group expressed a more restrictive attitude towards extramarital liaisons and evaluated their partner in more positively. As with SES 3 when SES 4 subscales were analysed by seizure type there were no significant differences.

Thus whatever the professed differences in sexual morality and feelings about psychosexual stimulation those women with epilepsy receiving therapy appeared to be

no different in their attitudes towards their marriage or in the strength of their sexual feelings towards their partners. Marital status can be used as measure of sexual interest, albeit a crude one, and examination of rates of marriage in the female group revealed no significant differences between the control, untreated or treated groups. Neither were there any significant differences between the seizure type groups and controls, with those women with CPS no less likely to be married than women suffering from other types of seizure. Most interesting was the finding that women developing epilepsy in the first decade were not significantly less likely to be married than those who sustained their first attack later in life, and that age at onset of epilepsy appeared not to influence AE (arousal by erotic imagery) scores suggesting that there is not a cumulative effect of seizure activity on the mechanisms of arousability.

Care must be taken in the interpretation of the results discussed above. The female control group was not well matched with the treated and untreated groups in that the control group, by the criteria employed, were much more likely to have received further education which is known to influence SES scores, and although the control and treated groups were well matched for age, the mean age of the untreated group was significantly lower than both controls and treated. Age can also affect SES scores although in these calculations of multiple regression it did not appear to exert a significant predictive effect. Some of the confidence intervals for SES subscale scores were wide and in some of the multiple regression there were large residuals both of which should add to the caution with which one interprets the results obtained.

For the women Spearman rank correlation of the number of seizures in the preceding 3 months and AE (arousal by erotic imagery) subscale of SES 2 score was not significant. There was a statistically significant value found for simple linear regression of seizure frequency in the preceding three months and FI (preferred frequency of intercourse) subscale scores but the correlation coefficient was modest and the R-sq (adj) = 11.7 % only. From these results it would appear that seizure frequency does not exert a strong influence on these aspects of sexual activity. AEDs which are known to affect cognitive function did not appear to affect AE scores and by implication the ability of the women studied to become aroused by erotic imagery.

Although the study design did not enable the author to make any comment on the effects if any of individual AEDs on SHBG, TT, FT, DHEAS, and androstenedione levels she did find that those women receiving treatment had higher SHBG and TT concentrations than those who were not. An observation that was first made almost twenty years ago in women with epilepsy. These changes not withstanding calculated FT was not significantly affected by AED therapy. An important finding given that it is the free moiety of testosterone which is physiologically active. Indeed those women receiving treatment exhibited a rise in midcycle FT levels suggesting that whatever effects AEDs are having on SHBG levels physiological concentrations of FT are being maintained.

The only significant relationship between FT and SES scores was a negative correlation between FI (preferred frequency of intercourse) and FT levels for those women in the treated group. Studies have shown that the relationship between androgens and sexual

appetite in women can be obscured by psychosocial circumstances to a greater extent than in men. There is also some suggestion in this literature that " too much " testosterone may by dint of enhancing a woman's libido make her more likely to take the initiative rendering her less attractive to her partner. Other factors which may outweigh FT levels are mood, general well being and feelings about the relationship. Ideally these topics should be examined along with questions pertaining to sexual function, and had we done so we may have found some explanation for this result.

The male treated group had a significantly higher mean SES 1 unweighted score than the control group, and when the subscales were calculated the treated group had a significantly higher mean MS (sexual morality within marriage) score compared with the controls, as with women multiple regression showed further education had a significant predictive effect. The confidence intervals were broad, however, and some care must be taken in interpreting these results. Both the untreated and treated groups had significantly higher mean unweighted SES 2 scores than the control suggesting and overall tendency to avoid psychosexual stimulation. The untreated group had a higher mean IS (interpersonal attraction) score than the control group, and both untreated and treated groups had higher mean AE (arousal by erotic imagery) scores than controls. These results suggest that some of the men with epilepsy reported a lower interpersonal attraction and found erotic love scenes etc less arousing than controls. Multiple regression showed that further education was a predictor for AE scores. As with the female sample men in the control group were much more likely to have received further education than the other groups, in addition the treated male group was significantly

older than both the untreated and controls a factor which Vennix and Frenken in their validation of the SES exerted a significant effect. This two factors must be borne in mind when interpreting these results. There were no significant differences between the three study groups for both SES 3 and SES 4 unweighted scores, neither were there any significant differences between the three study groups on calculation of the SES 3 subscales. Men receiving AEDs had a significantly higher lower MS (marital satisfaction) subscale score than either controls and untreated implying that they found their marriage more satisfying than the other two groups.

The male CPS group was significantly older than the control group, which should be borne in mind when looking at the SES results in relation to seizure type. The PGE and CPS groups had significantly higher MS (sexual morality within marriage) scores then the controls, and the PGE and undefined groups had significantly higher mean scores than controls on the AE (arousal by erotic imagery) subscale. Thus on the basis of these results there is little evidence of a deficit in arousability amongst men with CPS. There were no differences between the seizure type groups on SES 3, SES 4 and their attendant subscales. Neither were the men with CPS or any other form of epilepsy less likely to be married than the control group, although we did fine a phenomenon previously reported of men who developed seizures before puberty being significantly less likely to be married than those sustaining a first seizure thereafter. This observation could point to an element of sexual dimorphism or it could reflect differences in socialisation between the sexes. Lindsay's study was done on children developing epilepsy in the early sixties when there was still a tendency for children to be educated in

the different roles and behaviour expected of men and women. Boys would be expected to take the initiative and after marriage be the main breadwinner and leader of the household. Any ailment which undermined the self confidence necessary to undertake these tasks could well lead to an avoidance of intimacy with the opposite sex. The other explanation is that damage to the hippocampal-amygdalar complex at an early age in some way compromises the male's ability to "read" non - verbal signals from the female. Individuals of both sexes with bilateral amygdalar damage have difficulty in imagining and drawing faces expressing certain emotions, lesser unilateral damage may be enough to jeopardise recognition of some but not all types of facial expression. The fact that age at onset of epilepsy did not appear to affect AE (arousal by erotic imagery) may be explained by the fact that the erotic imagery enquired about was of a fairly explicit nature. As with the women AED levels did not affect AE (arousal by erotic imagery)

The design of the male arm of this study enabled the author to examine the effects of certain individual AEDs on SHBG, TT, calculated FT, AND, and DHEAS. Those men taking enzyme inducing AEDs and combinations of AEDs, at least one of which was usually an enzyme inducer, had higher SHBG and TT levels than controls, untreated and those in the VPA group. This is in line with previous studies and the reasons underlying this finding are discussed in the previous section. Of greater interest was the finding that calculated FT was not affect by AED therapy. Recent studies using direct measurement of FT have confirmed this. As FT is the physiologically active part of TT then it would appear that any defect in libido in men taking AEDs is not due to reduced FT levels.

Most of the studies of sexual function in people with epilepsy have been conducted on the more severely affected. Some of these studies were performed in residential institutions where access to the opposite sex let alone privacy, sex education, and contraception were presumably limited. The belief that offspring of people with epilepsy would also have the condition has been used in the past to discourage marriage, and by implication a full sexual life. In our society a person's self worth, especially a man's, has in the main been calculated in terms of the job he does and the salary earned, to say nothing of the type of car parked outside. Many people with epilepsy, no matter how well controlled, have had those opportunities denied them through ignorance on the part of the public at large. For those developing the disease in childhood the problems can be greater. The natural parental desire to protect the child may make the child overly dependant, unable to breakaway and forge their own adult relationships with both sexes. All of these factors have an effect on the individual psychosexual makeup, and are potentially compounded by the effects on cognition of repeated seizures, and AED therapy.

Despite all of these problems, the study population did not appear, beyond embracing a stricter moral code, and being less impressed by erotic imagery, to differ from the controls. Such differences as were found could well be explained by the discordance in age and further education between the controls and the study groups. Indeed those individuals with epilepsy seemed to have marginally more satisfactory marriages than the controls. There was no evidence of reduced FT levels in either sex making a purely hormonal explanation for reduced libido unlikely.

The essential difference between this study population, and that of some other groups was that all of the men and women who helped us were living independently in the community, and had gone to ordinary schools, thus their lives both at work and outside it were not so very different from their peers. Gore Vidal wrote that " sex is a matter of opportunity " it would appear from our findings that the men and women with epilepsy we studied were as keen to take their " opportunities " as the controls.

REFERENCES

ADAMS D.B., GOLD A.R., BURT A.D.
Rise in Female Initiated Sexual Activity at Ovulation and Its Suppression by Oral Contraceptives
New England Journal of Medicine 1978 299; 1145-1150

ANDERSON D. C.
Sex Hormone Binding Globulin
Clinical Endocrinology 1974 3 69-96

AKERT K GRUESEN A WOOLSEY C N MEYER D R
Kluver-Bucy Syndrome in Monkeys with Neocortical Ablations of Temporal Lobe
Brain 1961 84 480-497

ALSTROM C H
A Study of Epilepsy in its Clinical,Social and Genetic Aspects.
Acta Psychiatr Neurolo Scand 1950;63(Suppl):75-80

AMINOFF M F SIMON R P WIEDEMANN E
The Hormonal Responses To Generalized Tonic-Clonic Seizures
Brain 1984 107 569-578

BACKSTROM T. SODERGARD R.
Testosterone - Estradiol Binding Globulin, unbound and total estradiol and testosterone, and total progesterone during the menstrual cycle in women with epilepsy taking antiepileptic drugs
Journal of Endocrinological Investigation 1979 2 369 -366

BAIRD D.T., BURGER P.E., HEAVON-JONES G,D., SHARAMUZZI R.J.,
The site of production of androstenedione in non pregnant women
Journal of Endocrinology 1974 63 201-212

BALISH M ALBERT P S THEODORE W H
Seizure Frequency in Intractable Partial Epilepsy: A Statistical Analysis
Epilepsia 1991 32 642 - 649

BANCROFT J
Human Sexuality and its Problems
Churchill Livingston 1989

BANCROFT J., DAVIDSON D.W., WARNER P., TYRER G.
Androgens and Sexual Behavior in Women using Oral Contraceptives
Clinical Endocrinology 1980 12 327-340

BANCROFT J., WU F.C.W.
Changes in Erectile Responsiveness During Androgen Replacement Therapy.
Archives of Sexual Behaviour 1983 12 1 59-66

BANCROFT J., SHERWIN B.B., ALEXANDER G.M. DAVIDSON D.W., WALKER A.
Oral Contraceptives, Androgens, and the Sexuality of Young Women. II The Role of Androgens
Archives of Sexual Behavior 1991 20 121-135

BARRAGRY J M MAKIN H L J TRAFFORD D J H SCOTT D.F.
Effect of Anticonvulsants on Plasma Testosterone and Sex Hormone Binding Globulin Levels.
J.Neurol.Neurosurg. Psychiatry
41(1978) 913-941.

BEAR D LEVIN K BLUMER D CHETHAM D RYDER J
Interictal Behavior in Hospitalised Temporal Lobe Epileptics:Relationship to Idiopathic Psychiatric Syndromes.
J.N.N.P. 1982 45 481-488

BEAR D M FEDIO P
Quantative Analyis of Interictal Behavior in Temporal Lobe Epilepsy
Arch Neurol 1977 34 454-467

BERAN R G JENNINGS V R READ T
Doctors Perspectives of Epilepsy
Epilepsia 1981 22 397-406

BERAN R G READ T
A Survey of Doctors in Sydney,Australia:
Perspectives and Practicis Regarding Epilepsy and Those Affected by It.
Epilepsia 1983 24 79-104

BERGEN D DAUGHERTY S ECKENFELS E
Reduction of Sexual Actiivities in Females Taking Antiepileptic Drugs
Psychopathology 1992 25 1-4

BILLOD
Recherches et Considerations Relatives a la Symtomatologie de L'Epilepsie
Annales Medico - Psychologique 1843 381 -423

BILO L., MEO R., NAPPI C., ANNUNZIATO L., STRIANO S., COLAO A.M., MEROLA B., BUSCAINO A.
Reproductive Endocrine Disorders in Women with Primary Generalised Epilepsy.
Epilepsia 1988 29(5) 612-619

BLUMER D
Hypersexual Episodes in Temporal Lobe Epilepsy.
Amer.J Psychiat 1970 8 1099-1106

BLUMER D WALKER E
Sexual Behavior in Temporal Lobe Epilepsy
Arch Neurol 1967 16 37-43

BRODIE M J FREELY J
Adverse Drug Reactions
Br Med J 1988 296 845-849

BRODIE M J BOOBIS A R GILL M MASHITER K
Does Rifampicin Increase Serum Levels of Testosterone and Oestradiol by Inducing Sex Hormone Binding Globulin.
Br J Clin Pharmacol 1981 12 431-2

BROUGHTON R.J., GUBERMAN A., ROBERTS J.
Comparison of the Psychosocial Effects of Epilepsy and Narcolepsy/Cataplexy: A Controlled Study
Epilepsia 1984 25(4) 423-433

BRUTON C J
The Neuropathology of Temporal Lobe Epilepsy
Oxford University Press 1988

CALLAGHAN N O'HARE J O'DRISCOLL D O'NEILL DALY M
Comparitive Study of Ethosuxamide and Sodium Valproate in the Treatment of Typical Absence Seizures (Petit Mal)
Dev Med Child Neurol 1982 24 830-836

CAVENESS W F GALLUP G H
A Survey of Public Attitudes Towards Epilepsy in 1979 With an Indication of Trends Over the Past thirty Years.
Epilepsia 1980 21 509-518

a. CENDES F ANDERMAN F GLOOR P LOPES-CENDES I ANDERMANN E
MELANSON D JONES-GOTMAN M ROBITAILLE Y EVANS A PETERS T
Atrophy of Mesial Structures in Patients
with Temporal Lobe Epilepsy : Cause or Consequence of Repeated Seizures ?
Ann Neurol 1993 34 795-801

b. CENDES F ANDERMANN F GLOOR P EVANS A JONES-GOTMAN M WATSON C
MELANSON D OLIVIER A PETERS T LOPES-CENDES I LEROUX G.
MRI Volumetric Measurement of Amygdala and Hippocampus in Temporal Lobe Epilepsy.
Neurology 1993 43 719-725.

CHEN SHUN - SHENG MENG - RU SHEN TSAN-JU CHEN
SHUNG-LONG LAI
Effects of Antiepileptic Drugs on Sperm Motility of Normal Controls and Epileptic Patients with Long-Term Therapy.
Epilepsia 1992 33 149-153

CHENG R.W., REED M.J., JAMES V., T.,
Plasma Free Testosterone Equilibrium Dialysis Versus Direct Radioimmunoassay.
Clinical Chemistry 1986 32 1411-1415

CHRISTIANSEN P DEIGAARD J LUND M
Potency,Fertility,and Sexual Hormones in Young Male Epileptics.
Ugeskr Laeger 1975 137 2402-5

CLEMENT M J WALLACE S J
Juvenile Myoclonic Epilepsy
Arch Dis Child 1988 63 1049-1053

COGEN P. H., ANTUNES J. L., CORRELL J.W.,
Reproductive Function in Temporal Lobe Epilepsy: The Effect of Temporal Lobectomy
Surgical Neurology 1979 12 243-246.

CONNELL J M C RAPEPORT W G BEASTALL W G BRODIE M J
Changes in Circulating Androgens During Short Term Carbamazepine Therapy
Br.J Clin Pharmacol. 1984 17 347-58

CURRIER R.D., LITTLE S.C.,SUESS J. F., ANDY O. J.,
Sexual Seizures
Archives of Neurology 1971 25 260-264

DANA-HAERI J OXLEY J RICHENS A
A Reduction of Free Testosterone by Antiepileptic Drugs.
British Medical Journal 1982 284 85-86

DANA - HAERI J RICHENS A
Effect of Norethesterone on Seizures Associated with Menstruation
Epilepsia 1983 24 377 -381

DANA-HAERI J. TRIMBLE M.R., OXLEY J.
Prolactin and Gonadotrophin Changes Following Generalised and Partial Seizures.
Journal of Neurology, Neurosurgery, Psychiatry 1983 46 331-335

DANA-HAERI J OXLEY J RICHENS A
Pituitary Responsiveness to Gonadotrophin Releasing and Thyrotrophin
Hormones in Epileptic Patients Receiving Carbamazepine or Phenytoin.
Clinical Endocrinology(1984) 20 163-168

DANIELS O FENWICK P B C LELLIOTT P NDEGWA D WHEELER M NANJEE TOONE B.
Sex Hormone Replacement Therapy in Male Epileptics.
Advances in Epileptology XVth Epilepsy International Symposium.
ed.Porter R J et al 1984 291-297
Raven Press New York.

DANSKY L ANDERMANN E ANDERMANN F
Marriage and Fertility in Epileptic Patients.
Epilepsia 1980 21 261-271

DAVIES B M MORGENSTERN F S
a Case of Cystercosis,Temporal Lobe Epilepsy,and Transvestism.
J.Neurol.Neurosurg.Psychiat 1960 23 247-249

DECHAUD M., LEJEUNE H., GAROXIO-CHOLET M., MALLEIN R PUGEAT M.
Radioimmunoassay of Testosterone Not Bound to Sex Steroid Binding Protein in Plasma.
Clinical Chemistry 1989 35 1609-1614

DEMERDASH A., SHAALON M., MIDORI A.
Sexual Behaviour of a Sample of Females with Epilepsy
Epilepsia 1991 32 82-85

DESAI B. T., PORTER R. J., PENRY K.,
Psychogenic Seizures: A Study of 42 Attacks in Six Patients with Intensive Monitoring
Archives of Neurology 1982 39 202-209

DEWHURST K BEARD A W
Sudden Religious Conversion in Temporal Lobe Epilepsy.
Brit. J. Psychiat.1970 117 497-507

DICKS D MYERS R E KLING A
Uncus and Amygdala Lesions:Effects on Social Behavior in the Free-Ranging Rhesus Monkey.
Science 1969 69-71

DODRILL C B BREYER D N DIAMOND M B DUBINSKY B L GREARY B B
Psychosocial Problems Among Adults with Epilepsy.
Epilepsia 1984 25 168-175

DOWSETT M ATTREE S L VIRDEE S S DOWSETT M ATTREE S L
VIRDEE S JEFFCOATE S L
Oestrogen Related Changes in Sex Hormone Binding Globulin Levels During Normal and Gonadotrophin Stimulated Menstrual Cycles
Clinical Endocrinology 1985 23 303-312

DREIFUSS J.J., MURPHY J.T., GLOOR P.
Contrasting Effects of Two Identified Amygdaloid Efferent Pathways on Single Hypothalamic Neurons.
Journal of Neurophysiology 1986 31 237-248

DUCHARME J.R., COLLU R.
Pubertal Development: Normal, Precocious and Delayed.
Clinical Endocrinology and Metabolism 1982 11 57-87

EADIE M J
Anticonvulsant Drugs: An Update
Drugs 1984 27 328-363

EARLE K M BALDWIN M PENFIELD W
Incisural Sclerosis and Temporal Lobe Seizures Produced by Hippocampal Herniation at Birth
Arch Neurol Psych 1953 69 27-42

EPSTEIN A W
Relationship of Fetishism and Transvestism to Brain and Particularly to Temporal Lobe Dysfunction
J Nerv Ment Dis 1961 133 :247-253

FAIGLE J W FELDMANN K F
Carbamazepine Biotransformation
Antiepileptic Drugs p483 - 495 Eds. Woodbury DM, Penry JK, Pippenger CE
Raven Press New York 1982

FALCONER M A HILL D MYER A MITCHELL W POND D A
Treatment of Temporal-Lobe Epilepsy by
Temporal Lobectomy
Lancet 1955 23 827-835

FALCONER M A SERAFETINIDES E A NICHOLAS CORSELLIS J A
Etiology and Pathogenesis of Temporal Lobe Epilepsy.
Arch Neurol 1964 10 233-248

FALCONER M F TAYLOR D C
Surgical Treatment of Drug-Resistant Epilepsy Due to Mesial Temporal Sclerosis.
Arch Neurol 1969 19 353-360

FENWICK P B C MERCER S GRANT R WHEELER M NANJEE N TOONE B BROWN D.
Nocturnal Penile Tumescence and Serum Testosterone Levels.
Archives Sexual Behaviour 1986 15 13-21

FENWICK P B C TOONE B K WHEELER M J NANJEE M.N. GRANT R. BROWN D.
Sexual Behaviour in a Centre for Epilepsy
Acta.Neurol.Scand. 71 (1985) 428-435

FLOR-HENRY P.
Epilepsy and Psychopathology
Recent Advances in Clinical Psychiatry 1976
Ed. Granville-Grossman K. Churchill Livingstone Edinburgh

FRANCESCHI M PEREGO L CAVAGNINI F FRANCESCHI M PEREGO L CATTANE A G INVITTI C CAVIEZEL F STRAMBI L G SMIRNE S
Effects of Longterm Antiepileptic Therapy on the Hypothalamic - Pituitary Axis in Man.
Epilepsia 1984 25 46-52

FRANKS S., JACOBS H.S., MARTIN N., NABARRO J.D.N.
Hyperprolactinaemia and Impotence.
Clinical Endocrinology 1978 8 277-287

FREEMON F R NEVIS A H
Temporal Lobe Sexual Seizures.
Neurology 1969 19 87-90

FRENCH J A WILLIAMSON P D THADANI V M FRENCH J A WILLIAMSON P D THADANI V M DARCY T M MATTSON R H SPENCER S S
SPENCER D D
Characteristics of Medial Temporal Lobe Epilepsy 1: Results of History and Physical Examination
Ann Neurol 1993 34 774 - 778.

FRENKEN J VENNIX P
Sexuality Experience Scales
Netherlands Institute for Social Sexological Research (NISSO) Zeist The Netherlands
Swets & Zeitlinger BV 1981

GAGNON J SIMON W
Sexual Conduct:The Social Sources of Human Sexuality.
Aldine Chicago 1973.

GALBRAITH R.A., MICHNOVICZ J.J.
The Effects of Cimetidine on the Oxidative Metabolism of Estradiol.
New England Journal of Medicine
1989 321 269-274

GALLAGHER B.B., MURVIN A., FLANIGIN H. F., KING D.W., LUNEY D.
Pituitary and Adrenal Function in Epileptic Patients.
Epilepsia 1984 25(6) 683-689 1984

GASTAUT H COLLOMB H
Etude du Comportement Sexuel Chez Les Epileptiques Psychomoteurs.
Annales Medico-Psychologiques 1954 657-696

GAUTHIER-SMITH P C
Atteintes des Fonctions Cerebrales et Troubles du Comportement Sexuel.
Revue Neurologie(Paris) 1980 136 311-319

GILLHAM R.A., WILLIAMS N., WIEDMANN K.D. BUTLER E., LARKIN J.G. BRODIE M.J.
Cognitive Function in Adult Epileptic Patients Established on Anticonvulsant Monotherapy
Epilepsy Research 1990 7 219-225

GILLHAM R.A., WILLIAMS N., WIEDMANN K.
BUTLER E. LARKIN J.G. BRODIE M.J.
Concentration-effect relationships with carbamazepine and its epoxide on psychomotor and cognitive function in epileptic patients.
Journal Neurology, Neurosurgery and Psychiatry
1988 51 929-933

GLOOR P
Temporal Lobe Epilepsy: Its Possible Contribution to the Understanding of the Functional Significance of the Amygdala and of its Interaction with Neocortical-Temporal Mechanisms.
Neurobiology of the Amygdala
ED. Eleftheriou B E Plenum Press New York 1972.

GOEBELSMANN U ARCE J J THORNEYCROFT I H MISHELL D R
Serum Testosterone Concentrations in Women Throughout the Menstrual Cycle and Following HCG Administration
Am. J. Obstet. Gynecol.1974 445-452

GOFFMAN E
Stigma : Notes on the Management of Spoiled Identity
Prentice-Hall Inc 1963
Englewood Cliffs New Jersey USA

GOODWIN J SIMMS M BERGMAN R
Hysterical Seizures:A Sequel to Incest
Amer.J.Orthopsychiat.1979 49 698-703

GOOREN L.J.G.
Androgen Levels and Sex Functions in Testosterone Treated Hypogonadal Men
Archives of Sexual Behaviour 1987 (16) 6 463-473

GOWERS W R
Epilepsy and Other Chronic Convulsive Diseases. Their Causes, Symptoms and Treatment
William Wood New York 1885 p225

GRAM L.
Valproate, in Comprehensive Epileptology. Eds. Dram M., and Gram L.
1991 Raven Press New York (pages 537-546)

GREENAMYRE J T
The Role of Glutamate in Neurotransmission and in Neurological Disease
Arch Neurol 1986 43 1058 - 1063

GROSS M

Incestuous Rape:A Cause for Hysterical Seizures In Four Adolescent Girls.

Amer.J Orthopsychiat 1979 49 704-708

HANSSON R O DUFFIELD B J

Physical Attractiveness and the Attribution of Epilepsy

Journal Social Psychology 1976 99 233-240

HARTLAGE L C GREEN J B OFFUTT L

Dependancy in Epileptic Children

Epilepsia 1972 13 27-30

HERZOG A G

A Hypothesis to Integrate Partial Seizures of temporal Lobe Origen and Reproductive Endocrine Disorders

Epilepsy Research 1989 3 151-159

HERZOG A G

Intermittant Progesterone Therapy and Frequency of Complex Partial Seizures in Women with Menstrual Disorders

Neurology 1986 36 1607 - 1610

a. HERZOG A G SEIBEL M M SCHOMER D VAITUKAITIS J L GESCHWIND N

Reproductive Endocrine Disorders in Women with Partial Seizures of Temporal Lobe Origen

Arch Neurol 1986 43 341 - 346

HERZOG A G RUSSELL V VAITUKAITIS J L GESCHWIND N

Neuroendocrine Dysfunction in Temporal Lobe Epilepsy

Arch Neurol 1982 39 133-135

HERZOG A G LEVESQUE L A DRISLANE F W RONTHAL M.

SCHOMER D.L.

Phenytoin-Induced Elevation of Serum Estradiol and Reproductive Dysfunction in Men with Epilepsy

Epilepsia 32(1991) 550-553

b. HERZOG A G SEIBEL M M SCHOMER D L VAITUKAITIS J.L.

GESCHWIND N.

Reproductive Endocrine Disorders in Men with Partial Seizures of Temporal Lobe Origen.

Arch Neurol 1986 43 347-350.

HIERONS R SAUNDERS M
Impotence in Patients with Temporal Lobe Lesions.
Lancet 1966 2 761-764.

HILL D POND D A FALCONER M A
Personality Changes Following Temporal Lobectomy for Epilepsy
Journal of Mental Science 1957 103 18-27

HOARE P.
Psychiatric Disturbance in the Families of Epileptic Children
Developmental Medicine and Child Neurology 1984 26 14-19

HOARE P KERLEY S
Psychosocial Adjustment of Children with Chronic Epilepsy and Their Families.
Developmental Medicine and Child Neurology 1991 33 201-215

HOENIG J KENNA J.C.
EEG Abnormalities and Transsexualism
British Journal of Psychiatry 1979 134 293 - 300

HOOSHMAND H BRAWLEY B W
Temporal Lobe Seizures and Exhibitionism
Neurology 1969 19 1119-1124

HUNTER R LOGUE V MC MENEMY W H
Temporal Lobe Epilepsy Supervening on Longstanding Transvestism and Fetishism
Epilepsia 1963 4 60-65

ISMAIL A.A.A, ASTLEY P., CAWOOD M., SHORT F., WAKELIN K., WHEELER M.
Testosterone Assays : Guidlines for the Provision of a Clinical Biochemistry Service
Annals of Clinical Biochemisty 1986: 23 135-145

ISOJARVI J I T PAKARINEN A J MYLLYLA V V
A Prospective Study of Serum Sex Hormones During Carbamazepine Therapy.
Epilepsy Research 1991 9 139-144

ISOJARVI J I T MYLLYLA V V PAKARINEN A J
Effects of Carbamazipine on Pituitary
Responsiveness to Luteinizing Hormone Releasing Hormone,Thyrotropin Releasing Hormone and Metoclopramide in Epileptic Patients.
Epilepsia 1989 30 50-56

ISOJARVI J I T PAKARINEN A YLIPALOSAARI P.K. MYLLYLA V.V.
Serum Hormones in Male Epileptic Patients Receiving Anticonvulsant Medication.
Arch.Neurol. 47(1990) 670-676..

ISOJARVI J I T PAKARINEN A MYLLYA V V
Effects of Carbamazepine Therapy on Serum Sex Hormone Levels in Male Patients with Epilepsy
Epilepsia 29(1988) 781-786

JENSEN I LARSEN J K
Mental Aspects of Temporal Lobe Epilepsy
Follow up of 74 patients after resection a temporal lobe.
J.Neurol.Neurosurg.Psychiat.1979 42 256-265

JENSEN P., JENSEN S.B., SORENSEN P.S., BJERRE B.D., RIZZI D. A., SORENSEN A.S., KLYSNER R., BRINCH K., JESPERSEN B., NIELSEN H.
Sexual Dysfunction in Male and Female Patients With Epilepsy: A Study of 86 Outpatients.
Archives of Sexual Behaviour 1990 19 1 1-14

JOHNSON M H EVERITT B J
Essential Reproduction
Blackwell Scientific Publications
Oxford London Edinburgh Boston
Melbourne Paris Berlin Vienna 1991

JUDD H. L. YEN S.S.C.
Serum Androstenedione and Testosterone Levels During the Menstrual Cycle.
J Endocrinol Metab 1973 36 475-481

KIM M.H., ROSENFIELD R.L., DUPON C
The Effects of Dexamethasone on Plasma Free Androgens During the Normal Menstrual Cycle
American Journal of Obstetrics and Gynaecology 1976 126: 982-986

KLING A
Effects of Amygdalectomy and Testosterone on Sexual Behavior of Male Juvenile Macques.
Journal of Physiological Psychology 1968 65 466-470.

KLING A
Effects of Amygdalectomy on Social-Affective Behavior in Non-Human Primates
Neurobiology of the Amygdala
Ed. Eleftheriou B
Plenum Press New York 1972.

KLUVER H BUCY P C
Preliminary Analysis of Functions of the Temporal Lobes in Monkeys.
Archives of Neurology and Psychiatry
1939 42 979-1000.

KOLINARSKY A FREUND K MACHEK J POLAK O.
Male Sexual Deviation
Arch Gen Psychiat 1967 17 735-743

KRAEMER H C BECKER H B BRODIE H K H DOERING C H MOOS R H HAMBURG D A
Orgasmic Frequency and Plasma Testosterone in Normal Human Males.
Archives of Sexual Behaviour 1976 5 125-131

KWAN M., GREENLEAF W., J., MANN J., CRAPO L., DAVIDSON J.M.
The Nature of Androgen Action in Male Sexuality: A Combined Laboratory Self Report Study on Hypogonadal Men.
Journal of Clinical Endocrinology and Metabolism 1983 57 557-563

LANCET EDITORIAL
Sodium Valproate
Lancet 1988 1229-1231

LANGE J.D., BROWN W. A., WINCZE J.P., ZWICK W.
Serum Testosterone Concentration and Penile Tumescense Changes in Men
Hormones and Behaviour 1980 14 267-270

LECHTENBERG R
Epilepsy and the Family
Harvard University Press 1984

LEHMAN C
Epilepsy Association of America (Personal Communication) 1993

LELLY R CUMMINGS J L BENSON F FRANKEL M.
The Human Kluver Bucy Syndrome
Neurology(Cleveland) 1983 33 1141-1145

LENNOX W G MARKHAM C H
The Sociopsychological Treatment of Epilepsy.
J.A.M.A. 1953 1690-1694

LEPPIK I E
Carbamazepine
Comprehensive Epileptology p525-550 Eds. Dam M, Gram L
Raven Press New York 1990

LEVESQUE L.A. HERZOG A.G. SEIBEL M.M.
The effect of Phenytoin and Carbamezepine on Serum Dehydroepiandrosterone sulphate in Men and Women who Have Partial Seizures with Temporal Lobe Involvement.
Clinical Endocrinology and Metabolism 63 :243 1986.

LEVIN R. BANKS S. BERG B.
Psychosocial Dimensions of Epilepsy: A Review of the Literature.
Epilepsia 1988 29 805-816

LEVY R H KOCH K M
Drug Interactions With Valproic Acid
Drugs 1984 24 543-556

LILLY R., CUMMINGS J. L., BENSON F., FRANKEL M.
The Human Kluver - Bucy Syndrome
Neurology (Cleveland) 1983 33 1141-1145

LINDSAY J OUNSTED C RICHARDS P
Long Outcome in Children with Temporal Lobe Seizures I:Social Outcome and Childhood Factors.
Developmental Medicine and Child Neurology 1979 21 285-332

LISKE E., FORSTER F.M.,
Pseudoseizures: A Problem in the Diagnosis and Management of Epileptic Patients
Neurology 1964 8 41-49

LONG C G MOORE J R
Parental Expectation for Their Epileptic Children
J Child Psychol.Psychiat.1979 20 299-312

LUND L

Anticonvulsant Effect of Diphenylhydantoin Relative to Plasma level.A prospective Three Year Study in Ambulant Patients with Generalised Epileptic Seizures

Arch Neurol 1974 31 289 - 294

LUHDORF K., CHRISTIANSEN P., HANSEN J.M., LUND M.

The Influence of Phenytoin and Carbamazepine on Endocrine Function: Preliminary Results.

In Epilepsy VIII International Symposium Ed. Penry J.K. Raven Press New York 1977

MANYAM B V

Epilepsy in Ancient India

Epilepsia 1992 33 473-475

MATTHEWS W S BARABAL G FERRARI M

Emotional Concomitants of Childhood Epilepsy.

Epilepsia 1982 23 671-681

Mc INNES G.T., BRODIE M.J.

Drug Interactions That Matter - A Critical Reappraisal

Drugs 1988 36 83 -110

Mc PHEE G J A LARKIN J G BUTLER E BEASTALL G.H. BRODIE M.J.

Circulating Hormones and Pituitary Responsiveness in Young Epileptic Men Receiving Long-Term Antiepileptic Medication.

Epilepsia 29(1988) 468-475

McLEAN M J MacDONALD R L

Carbamazepine and 10,11 epoxycarbamazepine produce use and voltage dependant limitation of rapidly firing action potentials of mouse central neurons in cell culture

J Pharmacol Exp Ther 1986 238 727 - 738

MERRIT H H PUTNAM T J

Sodium Diphenyl Hydrantoinate in the Treatment of Convulsive Disorders.

J.Am Med Assoc 1938 111 1068 -1073

MINNS R A BROWN J K BLACKWOOD D H R McQUEEN J K

Valproate Levels in Children with Epilepsy

Lancet 1982 677-678

MITCHELL W. FALCONER M.A. HILL D.
Epilepsy With Fetishism Relieved by Temporal Lobectomy
Lancet 1954 2 626-630

MORRELL M.J., SPERLING M.R., STECKER M., DICHTER M.A.
Sexual Dysfunction in Partial Epilepsy: A deficit in physiologic sexual arousal
Neurology 1994 44 243- 247

MOTOHASHI T., WU C.H., ABDEL-RAHMAN M.A.,MARYMOR N. MIKHAIL G.
Estrogen/Androgen Balance in Health and Disease
American Journal of Obstetrics and Gynaecology 1979 135: 89-95

NANJEE M B WHEELER M J
Plasma Free Testosterone-Is an Index Sufficient ?
Ann Clin Biochem. 1985 22 387-90

NDEGWA D., RUST J., GOLOMBOK S, FENWICK P.
Sexual Problems in Epileptic Women
Sexual and Marital Therapy 1986 1 175-177

O'CARROLL R., SHAPIRO C., BANCROFT J.
Androgens, Behaviour and Nocturnal Erection in Hypogonadal Men: The Effects of Varying The Replacement Dose.
Clinical Endocrinology 1985 23 527-538

ODLIND V ELAMSSON K ENGLAND D E VICTOR A JOHANSSON E D B
Effects of Oestradiol on Sex Hormone Binding Globulin.
Acta Endocrinol 1982 101 248 - 253

PARRA A VELASCO M CERVANTES C MUNOZ H CERBON M A
VELASCO F
Plasma Prolactin Increase Following Electrical Stimulation of the Amygdala in Humans.
Neuroendocrinology 1980 31 60-65

PEARLMAN W.H., CREPY O., MURPHY M.
Testosterone Binding Levels in the Serum of Women During the Normal Menstrual Cycle, Pregnancy and Post-partum Period.
Journal of Clinical Endocrinology 1967 27 1012-1018.

PERSKY H., DRIESBACH L., MILLER W. R., O' BRIEN C. P.
The Relation of Plasma Androgen Levels to Sexual Behaviors and Attitudes of Women.
Psychosomatic Medicine 1982 44 305-319

PETRITZER B. K., FOSTER J.
A Case Study of A Male Transvestite with Epilepsy and Juvenile Diabetes
Journal of Nervous and Mental Disease 1955 121 557-563

PRITCHARD P B
Hyposexuality:A Complication of Complex Partial Epilepsy.
TRANSACTIONS OF THE AMERICAN NEUROLOGICAL ASSOCIATION. 1980 105:193-195

PRITCHARD P. B., WANNAMAKER B., SAGEL J., NAIR R., VILLIER C.
Endcrine Function Following Complex Partial Seizures.
Annals of Neurology 1983 4 27-32

PULLIANEN V. JOKELAINEN M.
Comparing the Cognitive Effects of Phenytoin and Carbamazepine in Long-term Monotherapy: A Two Year Follow-up.
Epilepsia 36 (12):1195-1202 1995

RAPEPORT W.G., MCINNES G.T., THOMPSON G.G.,FROREST G., PARK B.K., BRODIE M.J.
Hepatic Enzyme Induction and Leucocyte Delta Aminolaevulinic Acid Synthase Activity: Studies With Carbamazepine.
British Journal of Clinical Pharmacology 1983 16 133-137

RAO L.M., STEFAN H., BAUER J.
Epileptic But Not Psychogenic Seizures Are Accompanied by Simultaneous Elevation of Serum Pituitary Hormones and Cortisol Levels.
Neuroendocrinology 1989 49 33-39

RAUSCH R
Effects of Temporal Lobe Surgery on Behavior.
Advances in Neurology 1991 vol 55
Ed.Smith D,Treiman D,Trimble M.
Raven Press New York 1991

REMILLARD G.M. ANDERMANN F.,TESTA G.F., GLOOR P.,AUBE M., MARTIN J.B., FEINDEL W., GUBERMAN A., SIMPSON C.
Sexual Ictal Manifestations predominate in Women with Temporal Lobe Epilepsy: A Finding Suggesting Sexual Dimorphism in the Human Brain.
Neurology 1983 33 323 - 330

RENAUD L P
Influence of Amygdala Stimulation on the Activity of Identified Tuberoinfundibular Neurones in the Rat Hypothalamus.
J.Physiol 1976 260 237-252.

RODIN E SUBRAMANIAN M G GILROY J
Investigation of Sex Hormones in Male
Epileptic Patients.
Epilepsia 1984: 25(6) 690-694.

RODIN E SUBRAMANIAN M G SCHMALTZ S GILROY J.
Testosterone levels in Adult Male Epileptic Patients.
Neurology 1987 37 706-708

RUST J. GOLOMBOK S.
The GRISS : A Psychometric Instrument for the Assessment of Sexual Dysfunction.
Archives of Sexual Behaviour 1986 15 153-161

SAUNDERS M RAWSON M
Sexuality in Male Epileptics
J.Neurol.Sci 10 (1970) 577-583

SALMIMIES P, KOCKOTT G.,PIRKE K.M., VOGT H.J., SCHILL W.B.,
Effects of Testosterone Replacement in Hypogonadal Men
Archives of Sexual Behaviour (1982) 11 345-353

SELBY C

Sex Hormone Binding Globulin: Origen, Function and Clinical Significance.

Ann Clin Biochem 1990 27 532 - 541

SHERWIN B B

A Comparative Analysis of the Role of Androgen in Human Male and Female Sexual Behaviour: Behavioral Specificity,Critical Thresholds,and Sensitivity.

Psychobiology 1988 16 416-425

SHERWIN B B GELFAND M BRENDER W

Androgen Enhances Sexual Motivation In Females:A Prospective Study of Sex Steroid Administration in the Surgical Menopause

Psychosomatic Medicine 1985 47 339-351.

SHUKLA G D SRINVASTAVA O N KATIYAR B C

Sexual Disturbances in Temporal Lobe Epilepsy: A Controlled Study

Brit J Psychiat 1979 134 288-92

SIEVEKING E.H.

Analysis of 52 Cases of Epilepsy Observed by the Author.

Lancet 1857 (1) 527-528

SMALL J G MILSTEIN V STEVENS J R

Are Psychomotor Epileptics Different?

Archives Neurol 1962 7 188-194

SO E L KIFFEN PENRY J

Epilepsy in Adults

Ann Neurology 1981 9 3-16

SOUTHREN A L GORDON G G TOCHIMOTO S PINZON G LANE D R STYPULKOWSKI W

Mean Plasma concentration, Metabolic Clearance and Basal Plasma Production rates of Testosterone in Normal Young Men and Women Using a Constant infusion Proceedure Effect of Time of Day and Plasma concentration on the Metabolic Clearance Rate of Testosterone

Journal of Clinical Endocrin. and Metab 1967 27 686

SPARK R F WILLS C A ROYAL H

Hypogonadism,Hyperprolactinaemia and Temporal lobe Epilepsy in Hyposexual Men

Lancet 1 (Feb. 1984) 413-416

SPENCER S S SPENCER D D WILLIAMSON P D MATTSON R H

Sexual Automatisms in Complex Partial Seizures.

Neurology(Cleveland) 1983 33 527-533

SPERLING M R WILSON C L
The Effect of Limbic and Extralimbic Electical Stimulations Upon Prolactin Secretions in Humans
Brain Research 1986 371 293-297

STANDAGE D F
Etiology of Hysterical Seizures
Journal of Canadian Psychiatric Association 1975 20 67-73

STORES G
School-Children with Epilepsy at Risk for Learning and Behaviour Problems
Develop. Med. Child. Neurol. 1978 20 502-508

TAYLOR D C
Sexual Behaviour and Temporal Lobe Epilepsy
Archives of Neurology 1969 21 510-516

TAYLOR D C
Appetitive Inadequacy in the Sex behaviour of Temporal Lobe Epileptics
Journal of Neuro-Visceral Relations
1971 suppl x 486-490.

TEMKIN O
THE FALLING SICKNESS
A History of Epilepsy from the Greeks to the Beginnings of Modern Neurology
Second Edition 1971
John Hopkins Press Baltimore and London

TERZIAN H ORE G D
Syndrome of Kluver and Bucy Reproduced in Man by Bilateral Removal of Temporal Lobes.
Neurology 1953 5 373-380

TIZARD B
The Personality of Epileptics:A Discussion of The Evidence
Psychological Bulletin 1962 3 196-210

TOONE B. K., WHEELER M. FENWICK P.B.C.
Sex Hormone Changes in Male Epileptic Patients
Clinical Endocrinology 1980 12 391-395

TOONE B WHEELER M FENWICK P
Effects of Anticonvulsant Drugs on Male Sex Hormones and Sexual Arousal.
Psychopharmacology of Anticonvulsants
Oxford University Press 1982 Ed Sandler M 136-142

TOONE B WHEELER M NANJEE M FENWICK P GRANT R
Sex Hormones,Sexual Activity and Plasma Anticonvulsant Levels in Male Patients
J.Neurol.Neurosurgery and Psychiatry
1983 46 824-826

TOONE B
Sexual Disorders in Epilepsy.
Advances in Epilepsy 13
Ed.Pedlay T A Meldrum B S
Chuchill Livingston 1986

TOONE B K EDEH J FENWICK P GRANT R NANJEE N. PURCHES A.C. WHEELER M.

Hormonal and Behavioural Changes in Male Epileptics

Advances in Epileptology:XVth Epilepsy International Symposium.

Ed.Porter R.J.et.al. Raven Press New York 1984

TRIMBLE M R HERBERT J

The Effect of Testosterone or Oestradial upon the Sexual and Associated Behaviour of the Adult Female Rhesus Monkey.

J.Endocr. 1968 42 171-185

TRIMBLE M R

Serum Prolactin in Epilepsy and Hysteria

British Medical Journal 1978 ii 1682

TRIMBLE M R

Anticonvulsant Drugs and Cognitive Function: A Review of The Literature

Epilepsia 1987 28 s37-s45

UDRY J R TALBERT L M MORRIS N M

Biosocial Foundations for Adolescent Female Sexuality

Demography 1986 23 217-227

VELASCO M E TALEISNIK S

Release of Gonadotrophins Induced by Amygdaloid Stimulation in the Rat.

Endocrinology 1969 84 132-139

VERMEULEN A

The Androgens

Hormones in Blood 3rd edition 1979

Eds Gray C H James V H T pp 356-416

London Academic Press

VICTOR A LUNDBERG P O JOHANSSON E D B

Induction of Sex-hormone-binding-globulin by Phenytoin

Br.Med.Journal 1982 934

VON KRAFFT - EBBING R
Psychopathia Sexualis Trans. C.G.Chaddock
F.A.Davis Co.Philadelephia and London 1892

WADA J A ERIKSON L B
Menstrual Irregularities in Temporal Lobectomised Rhesus Monkeys(Macaca mulatta)
Science 1961 135 46-47

WARD F BOWER B D
A Study of Certain Social Aspects of Epilepsy in Childhood
Dev Med and Child Neurol Suppl 39 vol 20

WAXMAN S G GESCHWIND N
The Interictal Behaviour Syndrome of Temporal Lobe Epilepsy.
Arch.Gen Psychiatry 1975 32 1580-1586

WAXMAN S G GESCHWIND N
Hypergraphia in Temporal Lobe Epilepsy
Neurology 1974 24 629-636

WEBBER M P HAUSER W A OTTMAN R ANNEGERS F
Fertility in Persons with Epilepsy 1935-1974
Epilepsia 1986 27 246-252.

WILDER B J RAMSEY R E MURPHY J V KARAS B J MARQUARDT K HAMMOND E J
Comparison of Valproic Acid and Phenytoin in Newly Diagnosed Tonic- Clonic Seizures
Neurology 1983 33 1474-76

WILKE T.J., UTLEY D.J.
Total Testosterone, Free Androgen Index, Calculated Free Testosterone and Free Testosterone by Analogue RIA compared in Hirsute Women and in Otherwise Normal Women with Altered Binding of Sex Hormone Binding Globulin.
Clincal Chemistry 1987 33 1372-1375

WOOD C D
Behavioral Changes Following Discrete Lesions of Temporal Lobe Structures.
Neurology 1958 8 215-220

WOODBURY D.M.
Phenytoin: Proposed Mechanism of Anticonvulsant Action.
Antiepileptic Drugs: Mechanism of Action
Eds. Glaser G.H., Penry J.K., Woodbury D.M.
New York: Raven Press 1980 447-491

YAMADA Y GREER M A
The Effect of Bilateral Ablation of the Amygdala on Endocrine Function in the Rat.
Endocrinology 1960 66 565-570.

YERBY M S
Pregnancy and Teratogenesis Women and Epilepsy P167-192
Ed Trimble M R
John Wiley & Sons 1991

ZUCKERMAN M., LUBIN B., RINCK C.M.
Construction of New Scales for the Multiple Affect Adjective Check List
Journal of Behavioral Assessment 1983 5: 119-129

ZOLOVICK A.J.
Effects of Lesions and Electical Stimulation of the Amygdala on Hypothalamic-Hypophyseal - Regulation.
Neurobiology of the Amygdala
ED. Eleftheriou B E Plenum Press New York 1972.

APPENDICES

APPENDIX ONE : CONTROL GROUPS

FEMALE CONTROL GROUP

Nursing auxiliary	Accompanied patient to clinic
Housewife	Relative of Patient
Unemployed	Accompanied friend to clinic (no trade)
*Civil Servant	Recruited adjacent clinic (University)
*Hair Dresser	Accompanied sister to clinic (day release)
*Junior Doctor	Not known to investigators, working in orthopaedic clinic (University)
*secretary	Worked in Radiology (college)
*Nurse	Not known to investigators
*Secretary	Recruited adjacent clinic (college)
*Ward clerkess	Worked in female ward Department of Medicine (Not known to investigators)(day release)
*Technician	Recruited in outpatient corridor (college)
*Unemployed receptionist	Recruited in adjacent orthopaedic clinic (day release)
Shop assistant	Recruited in hospital shop adjacent to canteen(Not known to investigators)
Unemployed	Accompanied husband to clinic (no trade)
*Chiropodist	Recruited in main corridor (not known to investigators) (college)
Sales assistant	Recruited adjacent clinic
Auxiliary Nurse	Recruited in outpatients
*Housewife	Primary teacher with young children came to clinic to collect friend (college)

*Student Nurse	Working in orthopaedic clinic (college)
*Student	? Media studies. No further data
*Worked in bank	Accompanied son to clinic (Banking exams, day release)
*Pharmacy Technician	Recruited in outpatient corridor (not known to investigators) (college)
Hospital Cleaner	Recruited in outpatient corridor (Not known to investigators)
Hospital Seamstress	Not known to investigators
*Junior Doctor	Recruited in outpatient corridor (Not known to investigators)
*Florist	Recruited adjacent clinic (college)
Part time cleaner (not hospital employee)	Came to clinic with sister
*Secretary	Worked in local Building society. Recruited in WRVS coffee shop.(college)
*Librarian	Recruited in outpatient corridor (not known to investigators) (University)
*Director	Of family firm. Recruited in adjacent clinic (secretarial college)
*W.R.V.S Volunteer	Recruited in W.R.V.S canteen. Not known to investigators (secretarial college)
*Unemployed Nurse	Recruited adjacent clinic (college)
Shop assistant	Recruited adjacent clinic
*Teacher	Accompanied child to orthopaedic clinic (college)

*Secretary	Worked in hospital. Recruited outpatient corridor Not known to investigators (secretarial college)
*Hospital Hairdresser	Recruited in W.R.V.S canteen. Not known to investigators (secretarial college)
*Nun	Recruited in outpatient corridor. Not wearing habit (University)
Sales assistant	Accompanied boyfriend to clinic
*Psychologist	Visited epilepsy clinic as part of training (university)
Unemployed	Recruited in adjacent clinic (no trade)
Nursing auxiliary	Accompanied patient to clinic
*Care assistant in residential home	Accompanied patient to clinic
Auxiliary Nurse	Helped in Orthopaedic Clinic
*Hospital Receptionist	Worked in outpatients .Known to investigators (secretarial college)
Cleaner	Recruited in main corridor (not known to investigators)
*Student	Recruited in outpatient corridor (university).
*Postgraduate student	Recruited in corridor of department of medicine. Not known to investigators (University)

*Housewife

Adjacent clinic awaiting relative (Teacher training college):

* Denotes that individual claimed further education

MALE CONTROL GROUP

Hospital porter	Brought patient to clinic (not known to patient)
Hospital porter	Brought patient to clinic (not known to patient)
*Nurse	working in adjacent clinic (college)
*Hospital porter	Recruited in adjacent clinic (city and guilds printer)
*pharmacy technician	Recruited in outpatient corridor (college)
*Shipwright	Recruited in adjacent clinic (worked in Kvaerner/day release)
*Car mechanic	Recruited in adjacent clinic (apprenticeship/day release)
*Chemist	Came to collect friend form clinic (university)
Unemployed	Recruited in adjacent clinic (no trade)
Insurance Salesman	Recruited in adjacent clinic
*Assistant systems controller	Accompanied brother to clinic (university)
*Nurse	Recruited main corridor (unknown to investigators) (college)
*Primary teacher	Accompanied wife to clinic (college)
*Junior doctor	Working in adjacent orthopaedic clinic (not known to investigators)
*Student	Recruited in adjacent clinic (university)

*Junior doctor	Recruited in outpatient corridor (not known to investigators)
Clerical assistant	Recruited in adjacent clinic
Casual labourer	Sat in our waiting area believing we were the orthopaedic clinic
*Upholsterer	Accompanied patient to clinic (city and guilds)
*Joiner	Recruited in main corridor where he was working (not known to investigators) (city and guilds)
*Picture framer	Accompanied patient to clinic (day release at college)
Estate caretaker	Recruited in adjacent clinic
Local government officer	Recruited in adjacent clinic
*Consultant (Surgeon)	Recruited from orthopaedic clinic (not known to investigators)
*Fitter	Accompanied cousin to clinic (city and guilds)
*Plasterer	Recruited whilst refurbishing outpatient corridor(not known to investigators) (city and guilds)
Joiner	Recruited whilst refurbishing outpatient corridor(not known to investigators) (apprenticeship)

*Upholsterer	Accompanied mother to clinic (city and guilds)
Unemployed labourer	Recruited adjacent clinic
Physics technician	Recruited outpatient corridor (not known to investigators)
*Student	Recruited in adjacent clinic
Taxi driver	Came to pick up patient from clinic
Unemployed	Recruited in adjacent clinic
*Electician	Recruited whilst refurbishing outpatient corridor(not known to investigators) (city and guilds)
*Plumber	Recruited whilst refurbishing outpatient corridor(not known to investigators) (city and guilds)
*Pharmacist	Recruited in main corridor (not known to investigators) (university)
*Pathology technician	Recruited in W.R.V.S. canteen (not known to investigators) (college)
Porter	Brought specimens to clinic
*Medical student	Recruited in orthopaedic clinic (not known to investigators)
*Social worker	Accompanied patient to clinic (university)
Mortuary attendant	Recruited in main corridor
Unemployed school leaver	Accompanied father to clinic
Unemployed storeman	Relative of patient

*Lab. technician	Recruited in W.R.V.S. canteen (not known to investigators) (day release)
*Student nurse	Accompanied patient from ward to clinic

* Denotes that individual claimed further education

APPENDIX TWO : SES QUESTIONNAIRE

SES 1

By having pre-marital sexual intercourse you anticipate things, and that spoils much of the beauty of your marriage

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

I approve of children running around the beach in the nude.

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

For married people it is more important that the household runs smoothly than you are sexually well matched

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

When your're in love with each other premarital intercourse is perfectly natural

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

If you completely separate intercourse from childbearing it would soon turn into nothing but selfishness

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

When young people are told too much about sex they may easily go too far in experimenting with it

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

It is acceptable that an engaged couple has sex to -gether

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

Parents should forbid sexual games among young children

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

I could never be in love with a man / woman who already had alot of sexual experience

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

Before marriage it is your duty to your partner to maintain self control

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

In marriage it is more important that a man earns a good salary than he is sexually suited to his wife
Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

People should control themselves during orgasm (climax) and avoid groaning panting etc

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

The first time I had sex with a boy / girl it was with a boy / girl whom I

Knew only very superficially
Knew a little
Knew well
Was engaged to
Was married to
I have not had sex

I had premarital sexual contact (sexual intercourse)

Very often
Often
Now and then
Seldom
Never at all

I had premarital sexual contact (sexual intercourse)

None
Only my husband / wife
One or two others
Three to five others
Six or more others

A T.V. programme showing a naked man or woman should not be allowed even late in the evening.

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

SES 2

I find reading extensive descriptions of sex in novels

Very pleasant
Fairly pleasant
A little unpleasant
Very unpleasant

I could easily be aroused by a man / woman whom I knew has had a lot of sexual experience

Yes definitely
On the whole yes
Neutral
Not really
Not at all

I find watching T.V. shows with lots of nudity

Very pleasant
Fairly pleasant
A little unpleasant
Very unpleasant

I have sexual fantasies

Very Often
Often
Now and again
Seldom
Never at all

Flirting with nice men / women

I like alot
I like a little
I don't like
I strongly dislike

Do you like the idea that men / women notice you ?

Very much
A little
Not much
Not al all

I find indulging in sexual fantasies

Very pleasant
Fairly pleasant
A little unpleasant
Very unpleasant
If a movie showed all the details of sexual intercourse

I'd enjoy seeing it
I'd find it fairly pleasant
I'd find it a little unpleasant
I'd find it very unpleasant

While walking outside do you notice sexually attractive men / women ?

Very often
Often
Now and again
Seldom
Never at all

Are you sexually excited by erotic scenes in a romantic movie ?

Very Strongly
Strongly
Somewhat
Hardly
Not at all

Are you aroused by descriptions of sex in a novel ?

Very Strongly
Strongly
Somewhat
Hardly
Not at all

SES 4

How often do you think of partners other than your husband / wife during intercourse ?

Very often
Often
Now and then
Seldom
Never at all

When you are married there is nothing wrong in sometimes being in love with somebody else

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

" Free love " between man and woman would be preferable to the restrictions of married life

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

As far as sex is concerned every marriage becomes boring

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

When you are married it is your duty never to give into extramarital temptations

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

I can understand that over time people find marriage oppressive

Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

Are you satisfied with the affection your husband / wife shows ?

Not satisfied
Could be better
So so
Reasonably satisfied
Completely satisfied

Are you satisfied with the understanding your husband / wife shows for your problems and feelings

Not satisfied
Could be better
So so
Reasonably satisfied
Completely satisfied

If you could start all over again would you

Marry the same person ?
Marry someone else ?
Not marry

How often do you quarrel in your marriage compared with other couples you know?

Far more frequently
A little more frequently
As often
A little less frequently
Far less frequently

Are you sexually suited to your husband / wife ?

Very well couldn't be better
Reasonably well
So so
Could be better
Not very well

I find talking about our sexual relationship with my husband

Very easy couldn't be better
Reasonably easy
So so
Could be better
Not very easy

My husband / wife is sexually inhibited and prudish

Agree very strongly
Strongly
Somewhat
A little
Not at all

Are you satisfied with your sex life with your husband / wife ?

Not satisfied
Could be better
Reasonably satisfied
Completely satisfied

Since you got married have your feelings of affection and love for your husband / wife

Lessened sharply
Lessened somewhat
Remained the same
Increased somewhat
Increased strongly

How often in the last six months have you desired sexual intercourse with a man / woman other than your husband / wife ?

Very often
Often
Now and then
Seldom
Never at all

All in all how happy is your marriage ?

Extremely happy
Definitely happier than most
Somewhat happier than average
Definitely less happy than most
Very unhappy

How often have you regretted your marriage ?

Very often
Often
Now and then
Seldom
Never at all

My husband / wife wants sex too little

agree
Do not agree

My husband / wife wants sex too often

agree
Do not agree

(women only)
My husband doesn't do enough about sexual foreplay

agree
Do not agree

(Men only)
My wife shows a lack of enthusiasm for sex

agree
Do not agree

My husband / wife doesn't care enough about my own sexual wishes and preferences

agree
Do not agree

(Women only)
My husband often has difficulty in getting or keeping an erection during sex

agree
Do not agree

(Women only)
My husband reaches climax too quickly

agree
Do not agree

(Women only)
My husband is unable to reach a climax

agree
Do not agree
My husband wants to go to sleep or get up immediately after sex

agree
Do not agree

SES 3

Basically I find tongue kissing

Unpleasant
Not very pleasant
Fairly pleasant
Quite pleasant
Very pleasant

I find myself thinking I wish sex didn't exist

Very often
Often
Now and then
Seldom
Never at all

Do you become sexually aroused during petting or foreplay ?

Not at all
Hardly
Somewhat
Strongly
Very strongly

If my husband / wife didn't care about sex I would find it

Most unpleasant
Somewhat unpleasant
Not so bad
Not bad at all

Thoughts about sex frighten me a little

Very Often
Often
Now and then
Seldom
Never at all

I find lengthy or extensive foreplay

Very pleasant
Quite pleasant
Fairly pleasant
Not very pleasant
Unpleasant

I feel ill at ease sexually

Very strongly
Strongly
Somewhat
A little
Not at all

Our sexual foreplay is

Almost always short
Sometimes short, sometimes lengthy
Almost always lengthy

When my husband / wife caresses my body I find it

Unpleasant
Not very pleasant
Fairly pleasant
Quite pleasant
Very pleasant

I find touching my husband's / wife's genitals

Unpleasant
Not very pleasant
Fairly pleasant
Quite pleasant
Very pleasant

I have difficulty in getting sexually aroused during foreplay

Very often
Often
Now and then
Seldom
Never at all

How long on average do you think your sexual foreplay lasts on average before intercourse ?

2 minutes or less
3 - 5 minutes
6 - 10 minutes
11 - 15 minutes
16 -20 minutes
21 - 25 minutes
Greater than 25 minutes

How often do you prefer to have intercourse

Less than once a month
Once a month
2 - 3 times a month
Once a week
2 - 3 times a week
4 or more times a week

I am rather passive during intercourse

Very often
Often
Now and then
Seldom
Never at all

I feel somewhat listless and cold during intercourse

Very often
Often
Now and then
Seldom
Never at all

How do you like your husband being very active in intercourse ?

Very pleasant
Quite pleasant
Fairly pleasant
Not very pleasant
Unpleasant

I feel a little nervous and tense during intercourse

Very often
Often
Now and then
Seldom
Never at all

I find being completely naked during intercourse

Very pleasant
Quite pleasant
Fairly pleasant
Not very pleasant
Unpleasant

How do you like trying out different positions in intercourse

Very pleasant
Fairly pleasant
Somewhat pleasant
Very unpleasant

Do you have intercourse at times other than at night or in places other than in bed

Very often
Often
Now and then
Seldom
Never at all

(Women only)
I find caressing the male organ with my mouth

Very pleasant
Fairly pleasant
Slightly repulsive
very repulsive

How frequently have you had intercourse recently ?

Less than once per month
Once a month
2 -3 times a month
Once a week
2 - 3 times a week
Greater than 4 times a week

Generally speaking I find intercourse with my husband / wife
Very pleasant
Quite pleasant
Fairly pleasant
Not very pleasant
Unpleasant

On average how often do you get full orgasm during sexual intercourse ?

O out of 10
1 -2 times out of 10
3 - 5 times out of 10
6 - 7 times out of 10
8 - 9 times out of 10
10 times out of 10

Do you get full satisfaction from intercourse ?

Far too little
Not quite enough
Sufficient

I think most women need certain fantasies to reach full orgasm
Completely in agreement
On the whole in agreement
Neutral
Not really in agreement
Completely disagree

Do you get full satisfaction (climax) during intercourse ?

Never
Seldom
Now and then
Often
Usually always

I pretend to have a climax

Very often
Often
Now and then
Seldom
Never at all

After intercourse I feel somewhat let down

Very often
Often
Now and then
Seldom
Never at all

After sex how soon do you want sex again ?

In next hour
In same night
Next night
Within the next week

How often do you wake up feeling sexually aroused ?

Usually
Often
Now and then
Seldom
Never at all

(Men only)
How often do you have a morning erection ?

Usually
Often
Now and then
Seldom
Never at all

(Men only)
My wife cannot always reach climax

Usually
Often
Now and then
Seldom
Never at all

(Men only)
My wife reaches climax too slowly

Usually
Often
Now and then
Seldom
Never at all

(Men only)
Intercourse is often painful for my wife

Usually
Often
Now and then
Seldom
Never at all

Have you had any extra marital relationships ?

Yes
No

How many times have you had sex outside marriage ?

Weekly
Monthly
5-6 times per year
2-3 times per year
Less than once a year

APPENDIX THREE : NORMATIVE VALUES

Table 1:NORMATIVE VALUES FOR MALE SES SCALES (UNWEIGHTED)

	SCORE				
Percentile	**SES 1**	**SES 2**	**SES 3**	**SES 4**	**Percentile**
Hf	102	64	88	93	**Hf**
95-100	≥ 87	≥ 57	≥ 76	≥ 88	**95-100**
90-95	77/87	54/57	73/76	87/88	**90-95**
85-90	71/77	52/54	69/73	84/87	**85-90**
80-85	69/71	50/52	66/69	83/84	**80-85**
75-80	65/69	49/50	61/66	82/83	**75-80**
70-75	63/65	48/49	60/61	81/82	**70-75**
65-70	61/63	46/48	58/60	80/81	**65-70**
60-65	58/61	46	56/58	78/80	**60-65**
55-60	55/58	44/46	55/56	77/78	**55-60**
50-55	54/55	43/44	53/55	75/77	**50-55**
45-50	50/54	42/43	52/53	74/75	**45-50**
40-45	48/50	41/42	50/52	71/74	**40-45**
35-40	46/48	40/41	49/50	69/71	**35-40**
30-35	45/46	39/40	48/49	67/69	**30-35**
25-30	42/45	38/39	47/48	65/67	**25-30**
20-25	39/42	37/38	46/47	64/65	**20-25**
15-20	39	36/37	45/46	61/64	**15-20**
10-15	37/39	35/36	42/45	55/61	**10-15**
5-10	31/37	31/35	40/42	48/55	**5-10**
0-5	≤ 31	≤ 31	≤ 40	≤ 48	**0-5**
Lf	23	24	34	38	**Lf**
M	54.9	43.7	55.4	72.7	**M**
STD	16.6	7.9	11.4	11.5	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 243 Mean age = 37
M = mean STD = Standard Deviation

Table 2: VERBAL INTERPRETATION OF SES SCORES

SCORE MEN	SCORE WOMEN	MEANING
SES 1		
21- 48	21 - 50	Rejection of restrictive morality
49 - 80	51 73	Ambivalence re restrictive morality
81 - 105	74 - 105	Acceptance of restrictive morality
SES 2		
15 - 34	15 - 39	Strongly allowing psychosexual stimulation
35 - 43	40 - 51	Weakly allowing psychosexual stimulation
44 - 53	52 - 60	Weakly avoiding psychosexual stimulation
54 - 67	61 - 67	Strongly avoiding Psychosexual stimulation
SES 3		
29 - 60	29 - 70	Appetitive sexual motivation in interaction with partner
61 - 71	71 - 90	Sexual apathy in interaction with the partner
72 - 144	91 - 144	aversive sexual motivation in interaction with the partner
SES 4		
18 - 57	18 - 65	Attraction to marriage low
58 - 74	66 - 80	Attraction to marriage moderate
74 - 93	81 - 93	Attraction to marriage high

Table 3 :NORMATIVE VALUES FOR MALE SES 1 SUBSCALES (WEIGHTED)

	SCORE			
Percentile	**MS**	**PS**	**PA**	**Percentile**
Ht	2.00	1.35	1.63	**Ht**
Hf	1.96	1.35	1.63	**Hf**
95-100	≥ 1.24	≥ 1.35	≥ 1.44	**95-100**
90-95	0.83/1.24	1.35/1.35	1.19/1.44	**90-95**
85-90	0.67/0.83	1.25/1.35	0.91/1.19	**85-90**
80-85	0.51/0.67	0.80/1.25	0.71/0.91	**80-85**
75-80	0.33/0.51	0.51/0.80	0.44/0.71	**75-80**
70-75	0.22/0.33	0.40/0.51	0.31/0.44	**70-75**
65-70	0.12/0.22	0.28/0.40	0.17/0.31	**65-70**
60-65	0.06/0.12	0.20/0.28	0.11/0.17	**60-65**
55-60	-0.04/0.06	-0.01/0.20	-0.04/0.11	**55-60**
50-55	-0.14/-0.04	-0.03/-0.01	-0.14/-0.04	**50-55**
45-50	-0.25/-0.14	-0.18/-0.03	-0.27/-0.14	**45-50**
40-45	-0.33/-0.25	-0.24/-0.18	-0.37/0.27	**40-45**
35-40	-0.39/-0.33	-0.32/-0.24	-0.44/-0.37	**35-40**
30-35	-0.48/-0.39	-0.40/-0.32	-0.48/-0.44	**30-35**
25-30	-0.54/-0.48	-0.58/-0.40	-0.59/-0.48	**25-30**
20-25	-0.59/-0.54	-0.76/-0.58	-0.69/-0.59	**20-25**
15-20	-0.63/-0.59	-0.93/-0.76	-0.80/-0.69	**15-20**
10-15	-0.73/-0.63	-1.39/-0.93	-1.03/-0.80	**10-15**
5-10	-0.88/-0.73	-1.53/1.39	-1.03/-1.03	**5-10**
0-5	≤ -0.88	≤ -1.53	≤ -1.03	**0-5**
Lf	-0.96	-1.99	-1.03	**Lf**
Lt	-0.96	-1.99	-1.03	
M	-0.01	0.00	0.00	**M**
STD	0.65	0.91	0.75	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 236 Mean age = 37
M = mean STD = Standard Deviation

Table 4: NORMATIVE VALUES FOR MALE SES 2 SUBSCALES (WEIGHTED)

	SCORE			
Percentile	**IS**	**EE**	**AE**	**Percentile**
Ht	1.79	1.97	1.44	**Ht**
Hf	1.79	1.97	1.44	**Hf**
95-100	≥ 1.20	≥ 1.67	≥ 1.44	**95-100**
90-95	0.85/1.20	1.04/1.67	1.44/1.44	**90-95**
85-90	0.70/0.85	0.85/1.04	0.88/1.44	**85-90**
80-85	0.61/0.71	0.75/0.85	0.88/0.88	**80-85**
75-80	0.44/0.61	0.45/0.75	0.33/0.88	**75-80**
70-75	0.31/0.44	0.37/0.45	0.33/0.33	**70-75**
65-70	0.23/0.31	0.22/0.37	0.33/0.33	**65-70**
60-65	0.12/0.23	0.12/0.22	0.33/0.33	**60-65**
55-60	0.05/0.12	0.03/0.12	0.33/0.33	**55-60**
50-55	-0.04/0.05	-0.14/0.03	0.33/0.33	**50-55**
45-50	-0.11/-0.04	-0.26/-0.14	-0.23/0.33	**45-50**
40-45	-0.25/-0.11	-0.26/-0.26	-0.23/-0.23	**40-45**
35-40	-0.33/-0.25	-0.48/-0.26	-0.78/-0.23	**35-40**
30-35	-0.43/-0.33	-0.48/-0.48	-0.78/-0.78	**30-35**
25-30	-0.47/-0.43	-0.48/-0.48	-0.78/-0.78	**25-30**
20-25	-0.55/-0.47/	-0.56/-0.48	-0.78/-0.78	**20-25**
15-20	-0.65/-0.55	-0.78/-0.56	-0.78/-0.78	**15-20**
10-15	-0.82/-0.65	-1.11/-0.78	-1.34/-0.78	**10-15**
5-10	-1.12/-0.82	-1.48/-1.11	-1.89/-1.34	**5-10**
0-5	≤-1.12	≤-1.48	≤ -1.89	**0-5**
Lf	-1.74	-1.70	-3.00	**Lf**
Lt	-2.06	-1.70	-3.00	
M	0.00	0.00	-0.02	**M**
STD	0.68	0.87	0.97	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 236 Mean age = 37
M = mean STD = Standard Deviation

Table 5:NORMATIVE VALUES FOR MALE SES 3 SUBSCALES (WEIGHTED)

	SCORE				
Percentile	**EP**	**OE**	**II**	**FI**	**LF**
Ht	3.60	5.62	4.85	4.58	2.12
Hf	3.48	5.46	4.11	4.47	2.12
95-100	≥ 1.17	≥ 1.65	≥ 1.21	≥ 1.79	≥ 1.72
90-95	0.95/1.17	1.21/1.65	0.92/1.21	1.43/1.79	1.33/1.72
85-90	0.69/0.95	0.79/1.21	0.78/0.92	1.24/1.43	0.73/1.33
80-85	0.55/0.69	0.54/0.79	0.60/0.78	0.88/1.24	0.34/0.73
75-80	0.41/0.55	0.36/0.54	0.41/0.60	0.43/0.88	0.34/0.34
70-75	0.30/0.41	0.29/0.36	0.32/0.41	0.43/0.43	0.34/0.34
65-70	0.17/0.30	0.11/0.29	0.19/0.32	0.33/0.43	0.34/0.34
60-65	0.02/0.17	0.08/0.11	0.10/0.19	-0.03/0.33	0.34/0.34
55-60	-0.04/0.02	-0.10/0.08	0.01/0.10	-0.13/-0.03	-0.04/0.34
50-55	-0.13/-0.04	-0.14/-0.10	-0.12/0.01	-0.13/-0.13	-0.04/-0.04
45-50	-0.20/-0.13	-0.32/-0.14	-0.19/-0.12	-0.58/-0.13	-0.04/-0.04
40-45	-0.25/-0.20	-0.39/-0.32	-0.34/-0.19	-0.58/-0.58	-0.04/-0.04
35-40	-0.34/-0.25	-0.57/-0.39	-0.35/-0.34	-0.58/-0.58	-0.04/-0.04
30-35	-0.44/-0.34	-0.57/-0.57	-0.47/-0.35	-0.58/-0.58	-0.04/-0.04
25-30	-0.50/-0.44	-0.57/-0.57	-0.50/-0.47	-0.58/-0.58	-0.43/-0.04
20-25	-0.59/-0.50	-0.75/-0.57	-0.63/-0.50	-0.58/-0.58	-0.43/-0.43
15-20	-0.70/-0.59	-0.75/-0.75	-0.78/-0.63	-0.58/-0.58	-0.81/-0.43
10-15	-0.79/-0.70	-0.75/-0.75	-0.78/-0.78	-0.68/-0.58	-1.20/-0.81
5-10	-0.93/-0.79	-0.75/-0.75	-0.78/-0.78	-1.59/-0.68	-1.40/-1.20
0-5	≤-0.93	≤ -0.75	≤ -0.78	≤ -1.59	≤ -1.40
Lf	-1.14	-0.75	-0.78	-1.59	-2.19
Lt	-1.34	-0.75	-0.78	-1.59	-2.19
M	0.00	0.02	0.02	0.02	0.01
STD	0.72	0.97	0.72	1.01	0.87

Hf = Highest found score
Lf = Lowest found score
N = 236 Mean age = 37
M = mean STD = Standard Deviation

Table 6: NORMATIVE VALUES FOR MALE SES 4 SUBSCALES (WEIGHTED)

	SCORE			
Percentile	**MS**	**ES**	**AE**	**Percentile**
Ht	3.27	3.34	2.14	**Ht**
Hf	2.54	2.34	1.95	**Hf**
95-100	≥ 1.50	≥ 1.69	≥ 1.41	**95-100**
90-95	1.17/1.50	1.20/1.69	1.04/1.41	**90-95**
85-90	0.76/1.17	0.93/1.20	0.76/1.04	**85-90**
80-85	0.52/0.76	0.60/0.93	0.63/0.76	**80-85**
75-80	0.34/0.52	0.43/0.60	0.51/0.63	**75-80**
70-75	0.18/0.34	0.21/0.43	0.39/0.51	**70-75**
65-70	0.08/0.18	0.08/0.21	0.23/0.39	**65-70**
60-65	0.00/0.08	-0.04/0.08	0.13/0.23	**60-65**
55-60	-0.09/0.00	-0.12/-0.04	0.03/0.13	**55-60**
50-55	-0.14/-0.09	-0.19/-0.12	-0.04/0.03	**50-55**
45-50	-0.23/-0.14	-0.27/-0.19	-0.14/-0.04	**45-50**
40-45	-0.31/-0.23	-0.37/-0.27	-0.21/-0.14	**40-45**
35-40	-0.37/-0.31	-0.44/-0.37	-0.29/-0.21	**35-40**
30-35	-0.44/-0.37	-0.49/-0.44	-0.39/-0.29	**30-35**
25-30	-0.51/-0.44	-0.61/-0.49	-0.52/-0.39	**25-30**
20-25	-0.60/-0.51	-0.66/-0.61	-0.68/-0.52	**20-25**
15-20	-0.64/-0.60	-0.71/-0.66	-0.82/-0.68	**15-20**
10-15	-0.72/-0.64	-0.81/-0.71	-0.95/-0.82	**10-15**
5-10	-0.79/-0.72	-0.90/-0.81	-1.10/-0.95	**5-10**
0-5	≤ -0.79	≤ -0.90	≤ -1.10	**0-5**
Lf	-0.97	-0.90	-1.10	**Lf**
Lt	-0.97	-0.90	-1.10	
M	0.00	0.00	0.00	**M**
STD	0.72	0.77	0.73	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 236 Mean age = 37
M = mean STD = Standard Deviation

Table 7: NORMATIVE VALUES FOR FEMALE SES SCALES (UNWEIGHTED)

	SCORE				
Percentile	**SES 1**	**SES 2**	**SES 3**	**SES 4**	**Percentile**
Hf	99	67	123	93	**Hf**
95-100	≥ 87	≥ 61	≥ 97	≥ 90	**95-100**
90-95	84/87	60/61	90/97	89/90	**90-95**
85-90	80/84	59/60	84/90	88/89	**85-90**
80-85	77/80	57/59	81/84	86/88	**80-85**
75-80	73/77	56/57	79/81	85/86	**75-80**
70-75	71/73	55/56	77/79	84/85	**70-75**
65-70	69/71	54/55	73/77	82/84	**65-70**
60-65	66/69	53/54	71/73	81/82	**60-65**
55-60	63/66	52/53	69/71	79/81	**55-60**
50-55	62/63	51/52	67/69	79	**50-55**
45-50	59/62	50/51	65/67	78/79	**45-50**
40-45	57/59	48/50	62/65	77/78	**40-45**
35-40	53/57	47/48	60/62	75/77	**35-40**
30-35	50/53	46/47	59/60	74/75	**30-35**
25-30	47/50	45/46	57/59	71/74	**25-30**
20-25	45/47	44/45	55/57	68/71	**20-25**
15-20	40/45	42/44	52/55	66/68	**15-20**
10-15	37/40	41/42	48/52	63/66	**10-15**
5-10	33/37	37/41	44/48	54/63	**5-10**
0-5	≤ 33	≤ 37	≤ 44	≤ 54	**0-5**
Lf	24	16	36	38	**Lf**
M	60.6	50.1	68.2	76.7	**M**
STD	17.2	8.3	15.8	10.0	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 241 Mean age = 35
M = mean STD = Standard Deviation

Table 8:NORMATIVE VALUES FOR FEMALE SES 1 SUBSCALES (WEIGHTED)

	SCORE			
Percentile	**MS**	**PS**	**SS**	**Percentile**
Ht	1.64	1.42	1.13	**Ht**
Hf	1.51	1.42	1.13	**Hf**
95-100	≥ 1.05	≥ 1.25	≥ 1.13	**95-100**
90-95	0.95/1.05	1.07/1.25	0.90/1.13	**90-95**
85-90	0.79/0.95	0.93/1.07	0.74/0.90	**85-90**
80-85	0.61/0.79	0.81/0.93	0.62/0.74	**80-85**
75-80	0.50/0.61	0.65/0.81	0.53/0.62	**75-80**
70-75	0.37/0.50	0.47/0.65	0.44/0.53	**70-75**
65-70	0.27/0.37	0.36/0.47	0.38/0.44	**65-70**
60-65	0.21/0.27	0.19/0.36	0.30/0.38	**60-65**
55-60	0.10/0.21	0.11/0.19	0.20/0.30	**55-60**
50-55	0.02/0.10	-0.03/0.11	0.10/0.20	**50-55**
45-50	-0.10/0.02	-0.12/-0.03	-0.01/0.10	**45-50**
40-45	-0.19/-0.10	-0.30/-0.12	-0.13/-0.10	**40-45**
35-40	-0.35/-0.19	-0.40/-0.30	-0.28/-0.12	**35-40**
30-35	-0.48/-0.35	-0.54/-0.40	-0.39/-0.28	**30-35**
25-30	-0.56/-0.48	-0.70/-0.54	-0.49/-0.39	**25-30**
20-25	-0.67/-0.56	-0.78/-0.70	-0.75/-0.49	**20-25**
15-20	-0.82/-0.67	-0.93/-0.78	-0.96/-0.75	**15-20**
10-15	-0.90/-0.82	-1.00/-0.93	-1.24/-0.96	**10-15**
5-10	-1.07/-0.90	-1.14/-1.00	-1.65/-1.24	**5-10**
0-5	≤ -1.07	≤-1.14	≤ -1.65	**0-5**
Lf	-1.13	-1.58	-1.65	**Lf**
Lt	-1.13	-1.98	-1.65	
M	0.00	0.00	0.00	**M**
STD	0.68	0.78	0.77	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 239 Mean age = 35
M = mean STD = Standard Deviation

Table 9:NORMATIVE VALUES FOR FEMALE SES 2 SUBSCALES (WEIGHTED)

	SCORE			
Percentile	**IS**	**EE**	**AE**	**Percentile**
Ht	1.32	1.46	1.28	**Ht**
Hf	1.32	1.46	1.28	**Hf**
95-100	≥ 1.05	≥ 1.11	≥ 1.28	**95-100**
90-95	0.89/1.05	0.96/1.11	1.28/1.28	**90-95**
85-90	0.70/0.89	0.80/0.96	1.28/1.28	**85-90**
80-85	0.54/0.70	0.65/0.80	1.28/1.28	**80-85**
75-80	0.42/0.54	0.53/0.65	0.72/1.28	**75-80**
70-75	0.27/0.42	0.44/0.53	0.16/0.72	**70-75**
65-70	0.18/0.27	0.38/0.44	0.16/0.16	**65-70**
60-65	0.10/0.18	0.25/0.38	0.16/0.16	**60-65**
55-60	0.04/0.10	0.12/0.25	0.16/-0.16	**55-60**
50-55	-0.04/0.04	0.05/0.12	0.16/0.16	**50-55**
45-50	-0.11/-0.04	-0.06/0.05	0.16/0.16	**45-50**
40-45	-0.17/-0.11	-0.18/-0.06	0.16/0.16	**40-45**
35-40	-0.27/-0.17	-0.31/-0.18	-0.39/0.16	**35-40**
30-35	-0.39/-0.27	-0.44/-0.31	-0.39/-0.39	**30-35**
25-30	-0.51/-0.39	-0.57/-0.44	-0.95/-0.39	**25-30**
20-25	-0.61/-0.51	-0.68/-0.57	-0.95/-0.95	**20-25**
15-20	-0.80/-0.61	-0.83/-0.68	-0.95/-0.95	**15-20**
10-15	-0.96/-0.80	-0.96/-0.83	-0.95/-0.95	**10-15**
5-10	-1.24/-0.96	-1.56/-0.96	-1.50/-0.95	**5-10**
0-5	≤ -1.24	≤ -1.56	≤ -1.50	**0-5**
Lf	-2.72	-2.30	-3.17	**Lf**
Lt	-2.72	-2.30	-3.17	
M	-0.02	0.00	0.00	**M**
STD	0.70	0.78	0.95	**STD**

Hf = Highest found score
Lf = Lowest found score
N = 239 Mean age = 35
M = mean STD = Standard Deviation

Table 10: NORMATIVE VALUES FOR FEMALE SES 3 SUBSCALES (WEIGHTED)

Percentile	EP	OE	SI	FI	LF
Ht	2.65	2.44	3.28	4.36	2.08
Hf	2.58	2.44	3.28	4.18	2.08
95-100	≥ 1.33	≥ 1.68	≥ 1.16	≥ 1.64	≥ 1.66
90-95	1.01/1.33	1.24/1.68	0.98/1.16	1.31/1.64	1.24/1.66
85-90	0.70/1.01	0.96/1.24	0.76/0.98	0.88/1.31	0.66/1.24
80-85	0.57/0.70	0.80/0.96	0.62/0.76	0.60/0.88	0.66/0.66
75-80	0.39/0.57	0.67/0.80	0.47/0.62	0.36/0.60	0.24/0.66
70-75	0.30/0.39	0.42/0.67	0.29/0.47	0.26/0.36	0.24/0.24
65-70	0.23/0.30	0.16/0.42	0.16/0.29	0.26/0.26	0.24/0.24
60-65	0.17/0.23	0.00/0.16	0.06/0.16	0.16/0.26	0.24/0.24
55-60	0.04/0.17	-0.14/0.00	-0.02/0.06	0.02/0.16	0.24/0.24
50-55	-0.11/0.04	-0.30/-0.14	-0.12/-0.02	-0.01/0.02	0.24/0.24
45-50	-0.21/-0.11	-0.35/-0.30	-0.21/-0.12	-0.30/-0.01	-0.17/0.24
40-45	-0.29/-0.21	-0.36/-0.35	-0.26/-0.21	-0.49/-0.30	-0.17/-0.17
35-40	-0.37/-0.29	-0.46/-0.36	-0.34/-0.26	-0.60/-0.49	-0.17/-0.17
30-35	-0.50/-0.37	-0.54/-0.46	-0.43/-0.34	-0.61/-0.60	-0.17/-0.17
25-30	-0.58/-0.50	-0.64/-0.54	-0.53/-0.43	-0.84/-0.61	-0.17/-0.17
20-25	-0.65/-0.58	-0.67/-0.64	-0.67/-0.53	-0.84/-0.84	-0.59/-0.17
15-20	-0.72/-0.65	-0.81/-0.67	-0.81/-0.67	-0.84/-0.84	-0.70/-0.59
10-15	-0.87/-0.72	-0.88/-0.81	-0.87/-0.81	-0.84/-0.84	-1.27/-0.70
5-10	-1.00/-0.87	-1.18/-0.88	-0.98/-0.87	-1.18/-0.84	-1.60/-1.27
0-5	≤ -1.00	≤ -1.18	≤ -0.98	≤ -1.18	≤ -1.60
Lf	-1.27	-1.18	-1.12	-1.70	-2.42
Lt	-1.27	-1.18	-1.12	-1.70	-2.42
M	0.00	0.00	0.00	0.00	0.01
STD	0.71	0.85	0.74	0.95	0.90

Hf = Highest found score
Lf = Lowest found score
N = 239 Mean age = 35
M = mean STD = Standard Deviation

Table 11: NORMATIVE VALUES FOR FEMALE SES 4 SUBSCALES (WEIGHTED)

	SCORE			
Percentile	**MS**	**ES**	**AE**	**Percentile**
Ht	3.24	4.53	2.74	**Ht**
Hf	2.44	3.89	2.55	**Hf**
95-100	≥ 1.50	≥ 1.31	≥ 1.64	**95-100**
90-95	1.10/ 1.50	0.79/ 1.31	1.26/ 1.64	**90-95**
85-90	0.83/ 1.10	0.53/ 0.79	0.83/ 1.26	**85-90**
80-85	0.51/ 0.83	0.33/ 0.53	0.61/0.83	**80-85**
75-80	0.36/ 0.51	0.26/ 0.33	0.44/ 0.61	**75-80**
70-75	0.24/ 0.36	0.25/0.26	0.29/ 0.44	**70-75**
65-70	0.12/ 0.24	0.16/ 0.25	0.11/ 0.29	**65-70**
60-65	0.03/ 0.12	-0.02/ 0.16	0.01/ 0.11	**60-65**
55-60	-0.06/ 0.03	-0.11/ -0.02	-0.08/ -0.01	**55-60**
50-55	-0.13/-0.06	-0.26/ -0.11	-0.18/-0.08	**50-55**
45-50	-0.20/ -0.13	-0.27/ -0.26	-0.25/-0.18	**45-50**
40-45	-0.29/ -0.20	-0.36/ -0.27	-0.35/-0.25	**40-45**
35-40	-0.39/ -0.29	-0.53/ -0.36	-0.44/ -0.35	**35-40**
30-35	-0.47/ -0.39	-0.54/ -0.53	-0.55/ -0.44	**30-35**
25-30	-0.56/ -0.47	-0.54/ -0.54	-0.63/ -0.55	**25-30**
20-25	-0.63/ -0.56	-0.55/-0.54	-0.72/ -0.63	**20-25**
15-20	-0.68/ -0.63	-0.64/ -0.55	-0.72/ -0.72	**15-20**
10-15	-0.74/ -0.68	-0.64/-0.64	-0.81/-0.72	**10-15**
5-10	-0.84/ -0.74	-0.64/ -0.64	-0.81/ -0.81	**5-10**
0-5	≤ -0.84	≤ -0.64	≤ -0.81	**0-5**
Lf	-0.92	-0.64	-0.81	**Lf**
Lt	-0.92	-0.64	-0.81	
M	0.00	0.00	0.00	**M**
STD	0.72	0.71	0.77	**STD**

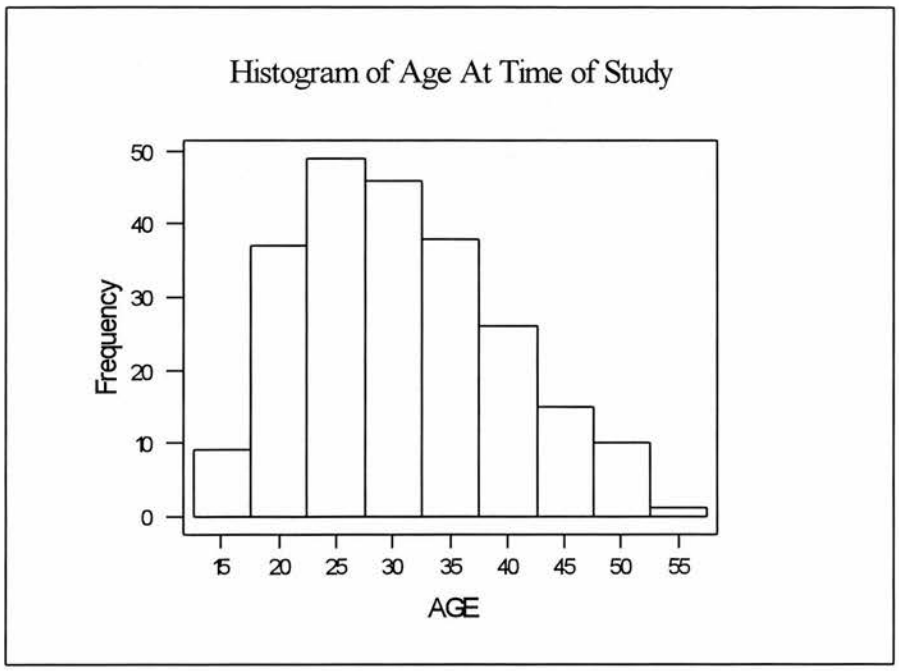
Hf = Highest found score
Lf = Lowest found score
N = 239 Mean age = 35
M = mean STD = Standard Deviation

APPENDIX FOUR : MALE STATISTICS

For multiple regression categorical data i.e. marital status, and whether a subject had received further education, was transformed into dummy variables such that those that were married were assigned the integer 1 whilst those that were not were assigned 0.Those who had received further education were assigned the value 1 those who had not 0.

MEN

Figure 1:ANOVA OF AGE AT TIME OF STUDY



Variable	N	Mean	Median	TrMean	StDev	SEMean
AGE	232	30.673	30.000	30.377	8.847	0.582

Variable	Min	Max	Q1	Q3
AGE	14.474	53.000	23.000	36.000

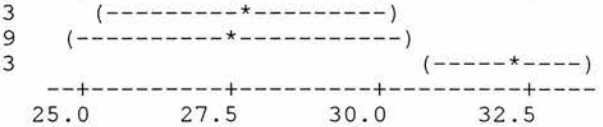
Analysis of Variance on AGE

Source	DF	SS	MS	F	p
GRP	2	1024.8	512.4	6.88	0.001
Error	228	16978.6	74.5		
Total	230	18003.4			

Level	N	Mean	StDev
1	45	27.800	7.143
2	34	27.620	8.119
3	153	32.176	9.113

Pooled StDev = 8.629

Individual 95% CIs For Mean Based on Pooled StDev

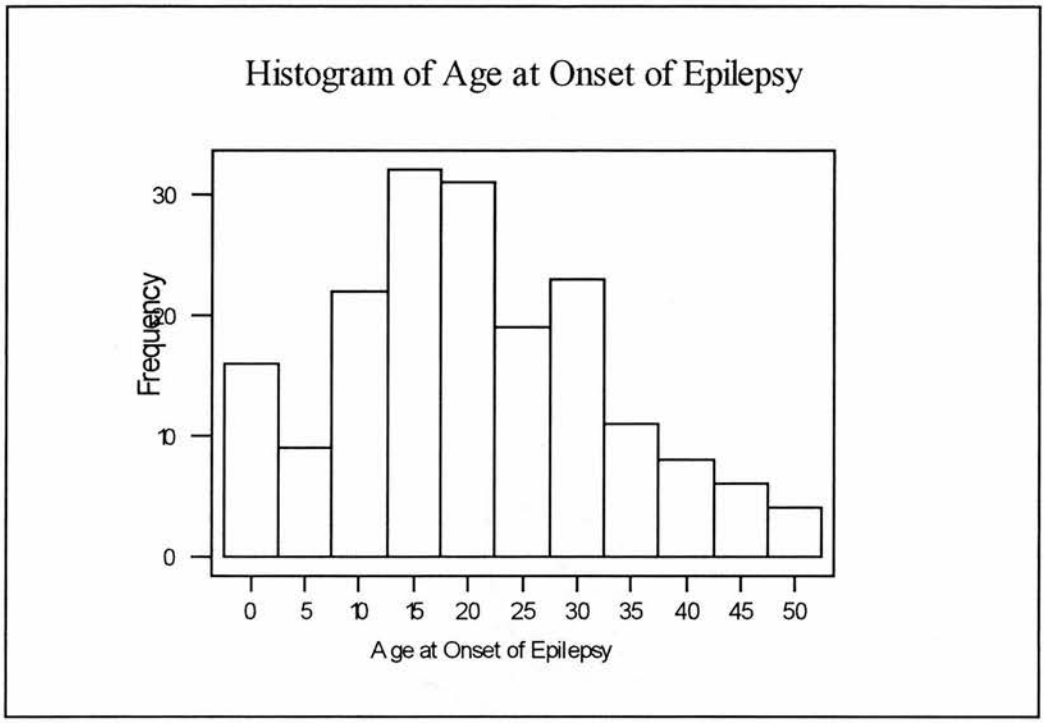


MEN

```
Fisher's pairwise comparisons

    Family error rate = 0.122
Individual error rate = 0.0500

Critical value = 1.970

Intervals for (column level mean) - (row level mean)

               1         2

     2    -3.717
           4.076

     3    -7.259    -7.819
          -1.494    -1.293
```

MEN

Figure 2:ANOVA OF AGE AT ONSET OF EPILEPSY



Variable	N	Mean	Median	TrMean	StDev	SEMean
AGE ONSET	187	20.474	19.000	20.109	12.100	0.899

Variable	Min	Max	Q1	Q3
AGE ONSET	0.000	52.000	12.000	28.000

Analysis of Variance

Source	DF	SS	MS	F	p
Factor	1	78	78	0.53	0.467
Error	185	26276	147		
Total	186	26354			

Level	N	Mean	StDev
C82	34	21.98	10.17
C83	153	20.19	12.44

Pooled StDev = 12.12

Individual 95% CIs For Mean Based on Pooled StDev

```
----------+---------+---------+------
(-----------------*-----------------)
   (-------*-------)
----------+---------+---------+------
        20.0      22.5      25.0
```

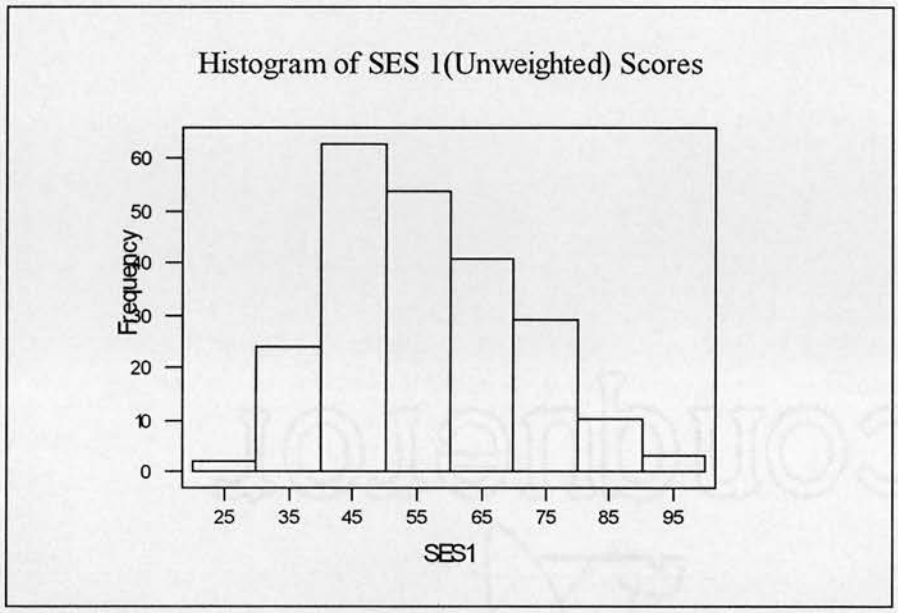
MEN

Figure 3:ANOVA OF AGES OF SEIZURE TYPE GROUPS

```
Analysis of Variance on AGE
Source     DF        SS        MS        F        p
seiztyp     3     918.0     306.0     4.01    0.009
Error     180   13735.9      76.3
Total     183   14653.9

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level      N      Mean     StDev  --------+---------+---------+--------
 CPS       75    33.941     9.105                    (------*------)
 PGE       86    30.471     9.714           (-----*-----)
 UND       26    30.444     7.006     (-----------*-----------)
 CONT      45    27.800     7.143  (------*-------)
                                      --------+---------+---------+--------
Pooled StDev =    8.736              28.0      31.5      35.0

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.973

Intervals for (column level mean) - (row level mean)

              1         2         3

     2     0.297
           6.643

     3    -1.228    -4.528
           8.222     4.582

     4     2.616    -0.622    -2.162
           9.666     5.965     7.451
```

MEN

Figure 4:ANOVA OF SES 1(Unweighted)



Variable	N	Mean	Median	TrMean	StDev	SEMean
SES1	232	55.704	53.000	55.333	13.924	0.926

Variable	Min	Max	Q1	Q3
SES1	26.000	96.000	45.000	66.000

Analysis of Variance on SES1

Source	DF	SS	MS	F	p
3GRP	2	2098	1049	5.63	0.004
Error	230	41523	186		
Total	231	43621			

Level	N	Mean	StDev
1	45	49.98	11.65
2	34	54.56	12.96
3	153	57.68	14.32

Pooled StDev = 13.65

Individual 95% CIs For Mean
Based on Pooled StDev

```
------+---------+---------+---------+
 (---------*---------)
           (----------*----------)
                        (----*-----)
------+---------+---------+---------+
    48.0      52.0      56.0      60.0
```

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

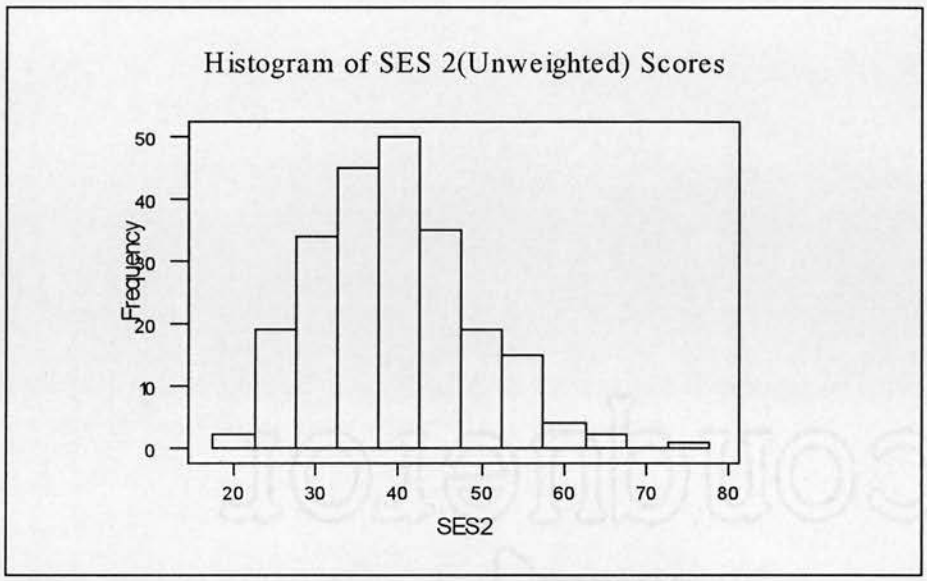
Critical value = 1.971

Intervals for (column level mean) - (row level mean)

	1	2
2	-10.80 1.63	
3	-12.27 -3.13	-8.36 2.12

MEN

Figure 5:ANOVA OF SES 2(Unweighted)



Variable	N	Mean	Median	TrMean	StDev	SEMean
SES2	232	39.500	39.000	39.201	9.205	0.612

Variable	Min	Max	Q1	Q3
SES2	19.000	73.000	33.000	45.000

Analysis of Variance on SES2

Source	DF	SS	MS	F	p
3GRP	2	957.7	478.9	5.90	0.003
Error	230	18108.8	81.2		
Total	231	19066.5			

Level	N	Mean	StDev
1	45	35.467	6.881
2	34	41.563	11.173
3	153	40.275	9.063

Pooled StDev = 9.011

Individual 95% CIs For Mean Based on Pooled StDev

```
-------+---------+---------+---------
 (------*-------)
                  (--------*--------)
                   (---*---)
-------+---------+---------+---------
    35.0      38.5      42.0
```

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

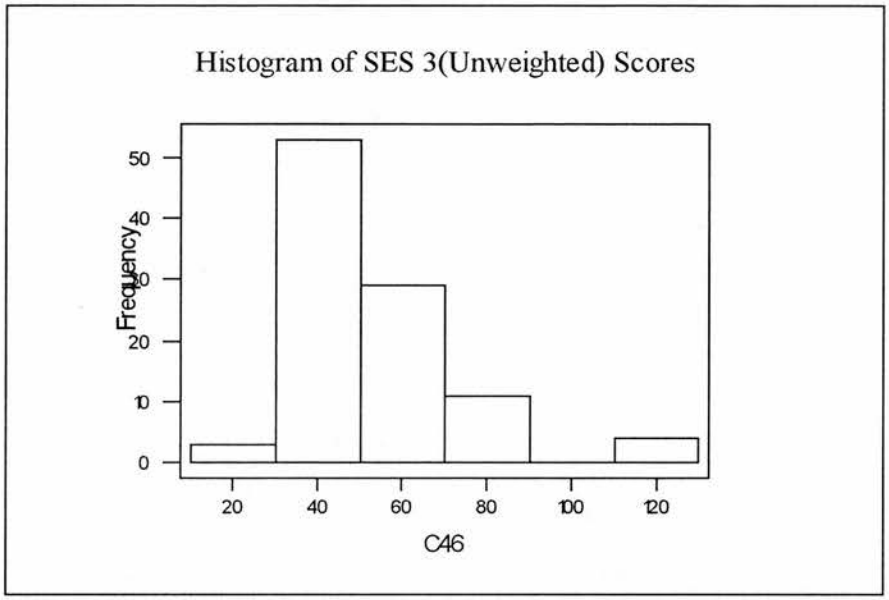
Critical value = 1.971

Intervals for (column level mean) - (row level mean)

	1	2
2	-10.203 -1.989	
3	-7.830 -1.787	-2.173 4.748

MEN

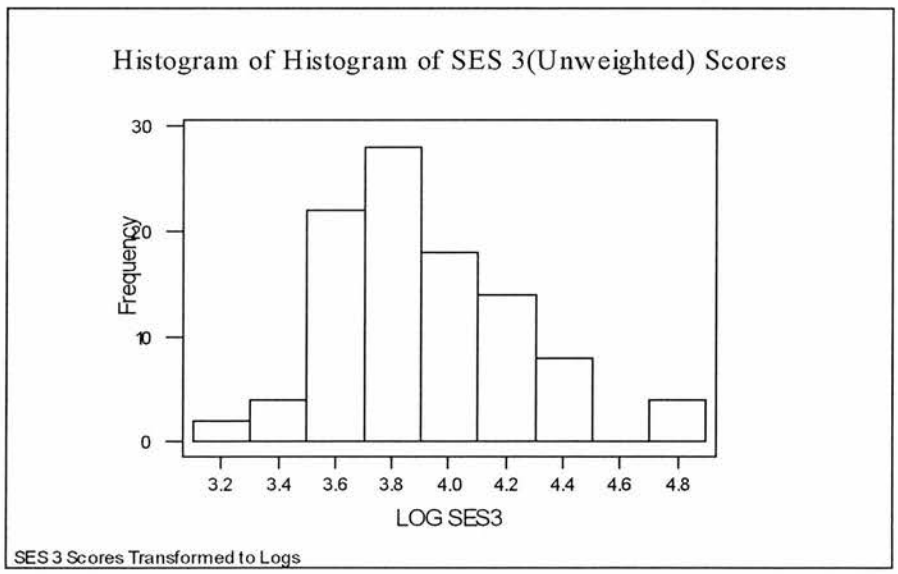
Figure 6:ANOVA OF SES 3(Unweighted)



Variable	N	Mean	Median	TrMean	StDev	SEMean
SES 3	102	52.44	47.00	50.59	18.76	1.87

Variable	Min	Max	Q1	Q3
SES3	27.00	124.00	39.50	59.00

SES 3 Scores not normally distributed. Logarithmic values taken



MEN

Figure 7:ANOVA OF SES 3(Unweighted){Logarithmic Values}

Analysis of Variance on LOG SES3

Source	DF	SS	MS	F	p
C47	2	0.314	0.157	1.51	0.226
Error	99	10.073	0.104		
Total	102	10.387			

Level	N	Mean	StDev
1	17	3.8496	0.3529
2	12	3.8086	0.1841
3	73	3.9537	0.3318

Pooled StDev = 0.3223

Individual 95% CIs For Mean
Based on Pooled StDev

```
---------+---------+---------+-------
        (------------*------------)
  (--------------*---------------)
                          (-----*------)
---------+---------+---------+-------
       3.72      3.84      3.96
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

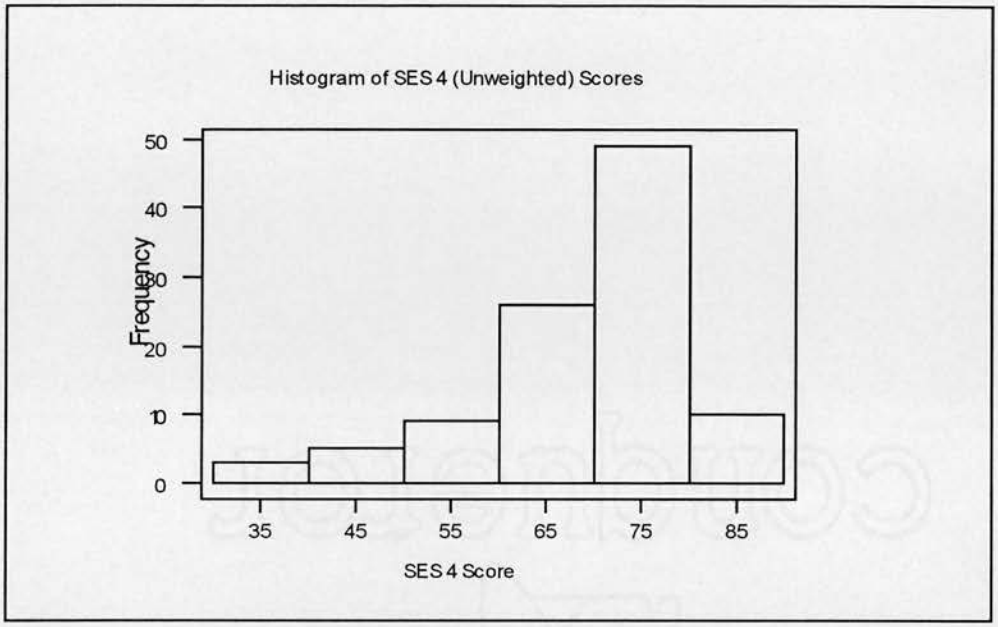
Critical value = 1.985

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.2002 0.2822	
3	-0.2768 0.0686	-0.3448 0.0545

MEN

Figure 8:ANOVA OF SES 4(Unweighted)



Variable	N	Mean	Median	TrMean	StDev	SEMean
SES4	102	68.06	70.00	68.93	10.71	1.06

Variable	Min	Max	Q1	Q3
SES4	33.00	84.00	64.75	75.25

Analysis of Variance on SES 4

Source	DF	SS	MS	F	p
C100	2	234	117	1.02	0.363
Error	100	11345	115		
Total	102	11580			

Level	N	Mean	StDev
1	17	70.18	6.52
2	12	70.83	12.42
3	73	67.11	11.16

Pooled StDev = 10.71

Individual 95% CIs For Mean Based on Pooled StDev

```
------+---------+---------+---------+
  (-------------*-------------)
 (----------------*-----------------)
 (------*------)
------+---------+---------+---------+
   66.5      70.0      73.5      77.0
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

Critical value = 1.984

Intervals for (column level mean) - (row level mean)

	1	2
2	-8.66 7.35	
3	-2.65 8.79	-2.89 10.34

MEN

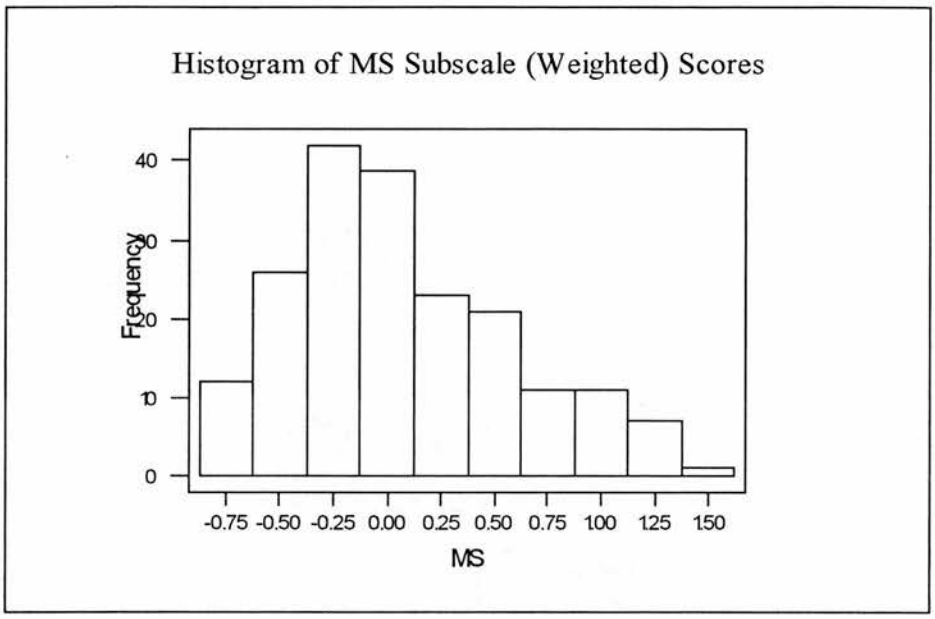
Because values not normally distributed the calculation was repeated using Kruskall-wallis.

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	18	71.00	55.3	0.58
2	12	73.00	65.5	1.74
3	73	70.00	48.3	-1.73
OVERALL	102		51.5	

H = 3.82 d.f. = 2 p = 0.149
H = 3.83 d.f. = 2 p = 0.148 (adjusted for ties)

MEN

Figure 9:ANOVA OF MS (Sexual Morality Within Marriage) SUBSCALE OF SES 1



Variable	N	Mean	Median	TrMean	StDev	SEMean
MS	232	0.0696	0.0000	0.0471	0.5170	0.0372

Variable	Min	Max	Q1	Q3
MS	-0.8300	1.5700	-0.3150	0.4200

Analysis of Variance on MS

Source	DF	SS	MS	F	p
3GRP	2	3.805	1.903	7.61	0.001
Error	229	47.516	0.250		
Total	231	51.322			

Level	N	Mean	StDev	Individual 95% CIs For Mean Based on Pooled StDev
1	45	-0.1793	0.4074	(---------*--------)
2	34	0.0231	0.4557	(----------*----------)
3	153	0.1663	0.5370	(----*-----)

Pooled StDev = 0.5001

-0.32 -0.16 0.00 0.16

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

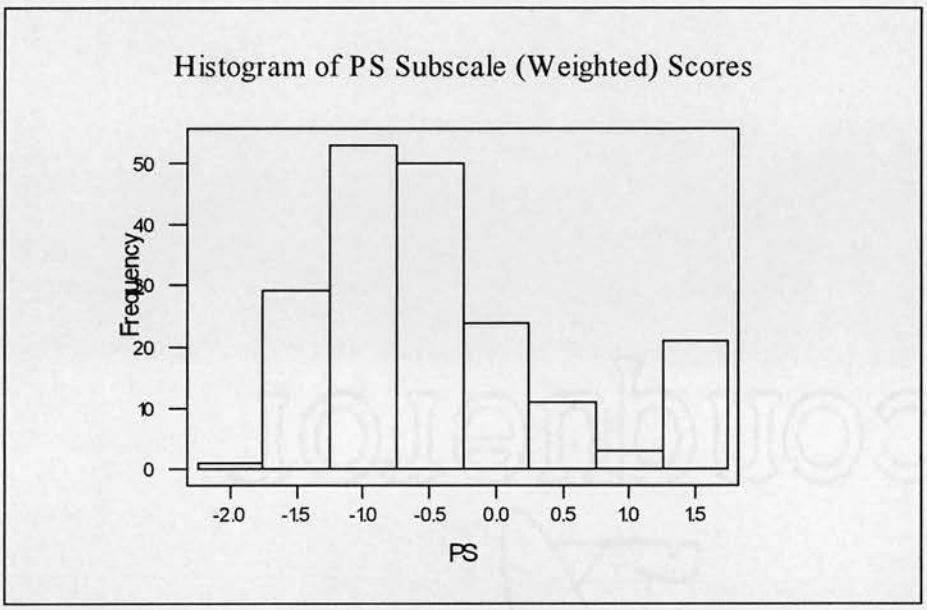
Critical value = 1.973

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.4406 0.0358	
3	-0.5221 -0.1691	-0.3470 0.0606

MEN

Figure 10:ANOVA OF PS (Premarital Sexual Experience) SUBSCALE OF SES 1



Variable	N	Mean	Median	TrMean	StDev	SEMean
PS	232	-0.4485	-0.6000	-0.4980	0.9176	0.0662

Variable	Min	Max	Q1	Q3
PS	-1.9700	1.6400	-1.1625	-0.0850

Analysis of Variance on PS

Source	DF	SS	MS	F	p
GRP	2	0.027	0.013	0.02	0.984
Error	229	160.789	0.851		
Total	231	160.816			

Level	N	Mean	StDev
1	45	-0.4640	0.9986
2	34	-0.4629	0.8102
3	153	-0.4392	0.9215

Pooled StDev = 0.9224

Individual 95% CIs For Mean Based on Pooled StDev

```
---------+---------+---------+------
   (-------------*-------------)
 (---------------*---------------)
           (-------*-------)
---------+---------+---------+------
      -0.60     -0.40     -0.20
```

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

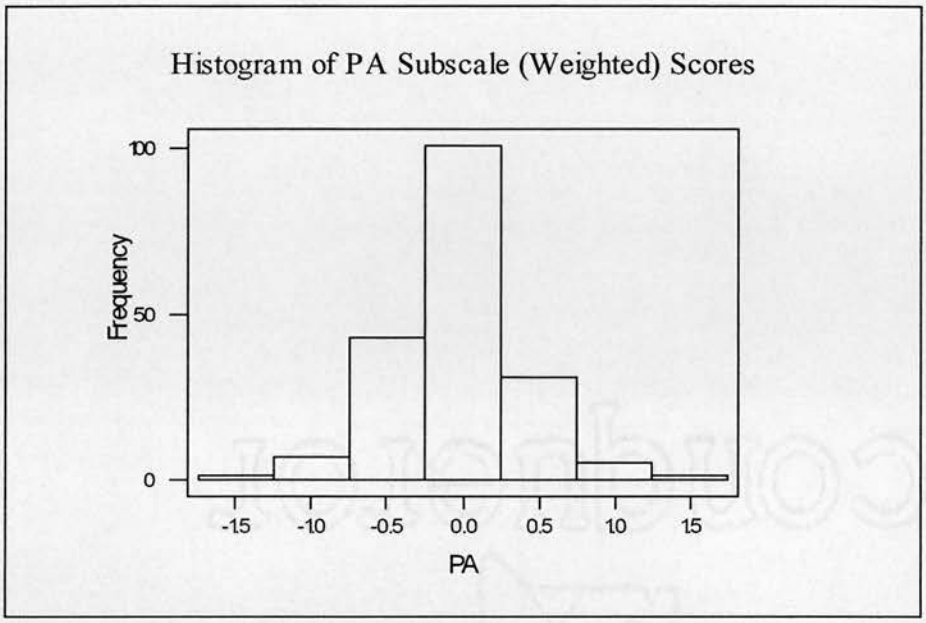
Critical value = 1.973

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.4320 0.4298	
3	-0.3514 0.3018	-0.3906 0.3433

MEN

Figure 11:ANOVA OF PA (Premarital Sexual Attitude) SUBSCALE OF SES 1



Variable	N	Mean	Median	TrMean	StDev	SEMean
PA	232	-0.0453	-0.0500	-0.0479	0.4001	0.0291

Variable	Min	Max	Q1	Q3
PA	-1.2700	1.6400	-0.2900	0.2100

Analysis of Variance on PA

Source	DF	SS	MS	F	p
3GRP	2	0.133	0.066	0.41	0.663
Error	229	29.956	0.161		
Total	231	30.089			

Level	N	Mean	StDev
1	45	-0.0388	0.3422
2	34	0.0143	0.4494
3	153	-0.0615	0.4081

Pooled StDev = 0.4013

Individual 95% CIs For Mean Based on Pooled StDev

```
-------+---------+---------+---------
 (-----------*-----------)
   (--------------*--------------)
    (------*------)
-------+---------+---------+---------
    -0.10      0.00      0.10
```

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

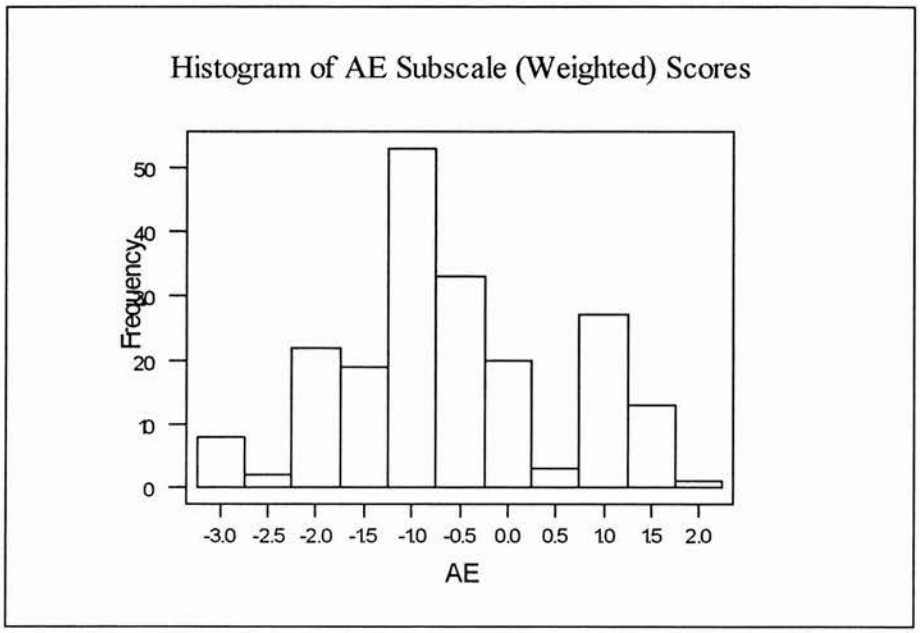
Critical value = 1.973

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.2472 0.1411	
3	-0.1205 0.1660	-0.0904 0.2420

MEN

Figure 12:ANOVA OF AE (Arousal by Erotic Imagery) SUBSCALE OF SES 2



Variable	N	Mean	Median	TrMean	StDev	SEMean
AE	232	-0.5430	-0.7800	-0.5171	1.0757	0.0759

Variable	Min	Max	Q1	Q3
AE	-3.0300	1.8500	-1.3650	0.2200

Analysis of Variance on AE

Source	DF	SS	MS	F	p
GRP	2	7.39	3.69	3.27	0.040
Error	229	224.05	1.13		
Total	231	231.44			

Level	N	Mean	StDev
1	45	-0.927	0.965
2	34	-0.417	1.067
3	153	-0.455	1.091

Pooled StDev = 1.064

Individual 95% CIs For Mean Based on Pooled StDev

```
------+---------+---------+---------+
(---------*--------)
              (----------*----------)
                   (----*----)
------+---------+---------+---------+
   -1.05     -0.70     -0.35     -0.00
```

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

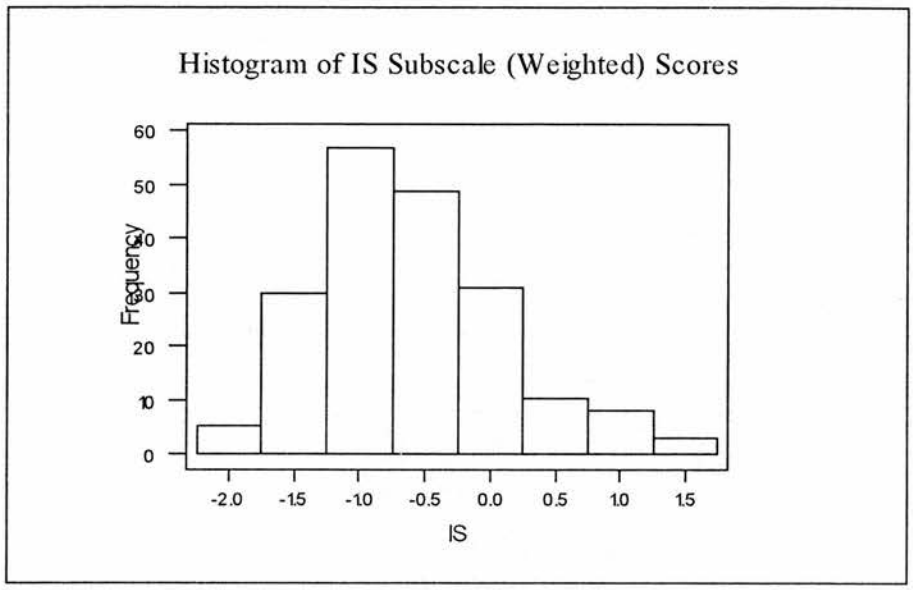
Critical value = 1.972

Intervals for (column level mean) - (row level mean)

	1	2
2	-1.017 -0.003	
3	-0.851 -0.093	-0.387 0.463

MEN

Figure 13:ANOVA OF IS (Interpersonal Sexual Attraction) SUBSCALE OF SES 2



Variable	N	Mean	Median	TrMean	StDev	SEMean
IS	232	-0.6296	-0.7200	-0.6597	0.7014	0.0505

Variable	Min	Max	Q1	Q3
IS	-2.0600	1.5100	-1.0950	-0.2300

Analysis of Variance on IS

Source	DF	SS	MS	F	p
3GRP	2	3.573	1.786	3.74	0.026
Error	229	90.872	0.478		
Total	231	94.445			

Level	N	Mean	StDev	Individual 95% CIs For Mean Based on Pooled StDev
1	45	-0.8317	0.5590	(--------*-------)
2	34	-0.3813	0.8099	(---------*---------)
3	153	-0.6211	0.7014	(----*----)

Pooled StDev = 0.6916 -1.00 -0.75 -0.50 -0.25

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

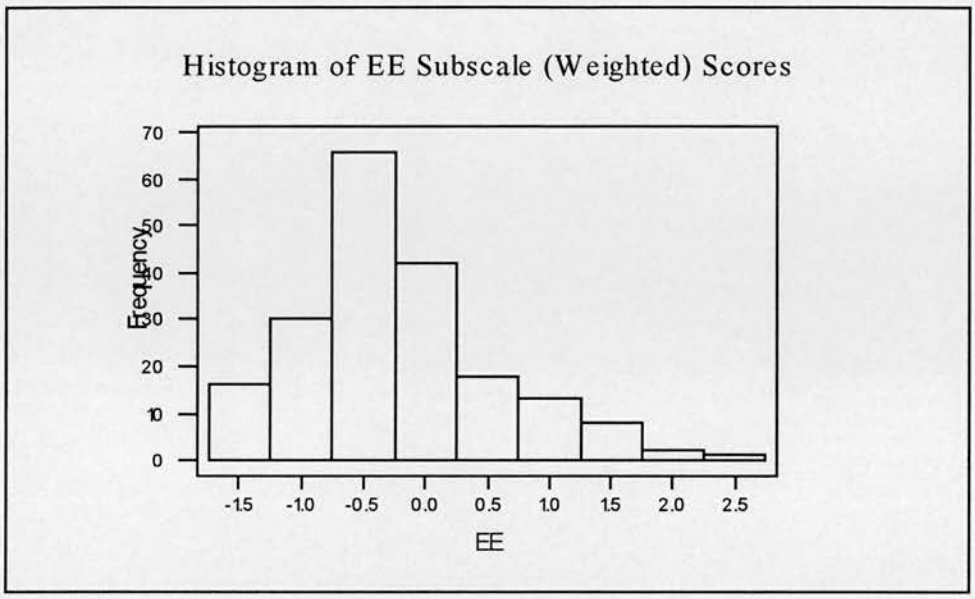
Critical value = 1.973

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.7765 -0.1242	
3	-0.4550 0.0338	-0.0386 0.5180

MEN

Figure 14:ANOVA OF EE (Evaluation of Exposure to Erotic Imagery) SUBSCALE OF SES 2



Variable	N	Mean	Median	TrMean	StDev	SEMean
EE	232	-0.2815	-0.4700	-0.3215	0.7638	0.0546

Variable	Min	Max	Q1	Q3
EE	-1.6900	2.4000	-0.6900	0.1200

Analysis of Variance on EE

Source	DF	SS	MS	F	p
3GRP	2	2.528	1.264	2.19	0.114
Error	229	111.228	0.576		
Total	231	113.756			

Level	N	Mean	StDev
1	45	-0.3837	0.6226
2	34	-0.0287	0.7567
3	153	-0.3098	0.8017

Pooled StDev = 0.7592

Individual 95% CIs For Mean
Based on Pooled StDev

```
-----+---------+---------+---------+-
 (--------*--------)
          (----------*----------)
       (-----*----)
-----+---------+---------+---------+-
  -0.50     -0.25      0.00      0.25
```

Fisher's pairwise comparisons

Family error rate = 0.122
Individual error rate = 0.0500

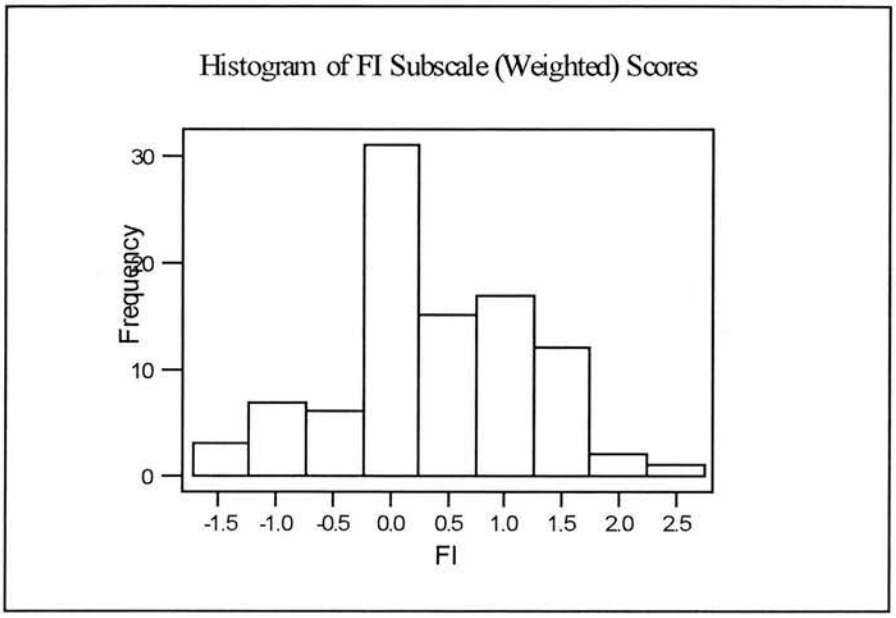
Critical value = 1.972

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.7077 -0.0023	
3	-0.3395 0.1915	-0.0201 0.5822

MEN

Figure 15:ANOVA OF FI (Preferred Frequency of Intercourse) SUBSCALE OF SES 3



Variable	N	Mean	Median	TrMean	StDev	SEMean
FI	103	0.2472	0.2180	0.2514	0.8651	0.0928

Variable	Min	Max	Q1	Q3
FI	-1.5900	2.4300	-0.2300	0.7700

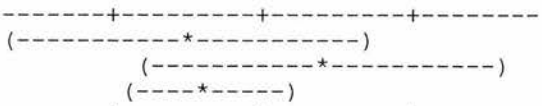
Analysis of Variance on FI

Source	DF	SS	MS	F	p
3GRP	2	1.112	0.556	0.77	0.469
Error	100	58.863	0.727		
Total	101	59.976			

Level	N	Mean	StDev
1	17	0.1990	0.7411
2	12	0.5655	0.9455
3	73	0.2486	0.8536

Pooled StDev = 0.8525

Individual 95% CIs For Mean Based on Pooled StDev



Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

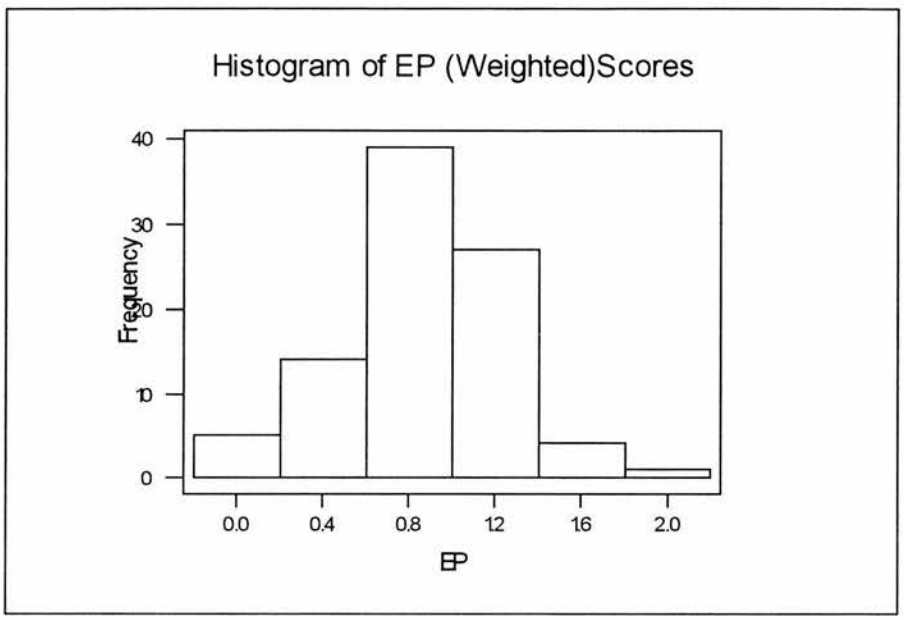
Critical value = 1.990

Intervals for (column level mean) - (row level mean)

	1	2
2	-1.0591 0.3261	
3	-0.5860 0.4869	-0.2195 0.8534

MEN

Figure 16:ANOVA OF EP (Enjoyment Potential of Sexual Interaction) SUBSCALE OF SES 3



Variable	N	Mean	Median	TrMean	StDev	SEMean
EP	102	0.9006	0.9300	0.8982	0.3552	0.0400

Variable	Min	Max	Q1	Q3
EP	0.0700	2.1800	0.6900	1.1200

Analysis of Variance on EP

Source	DF	SS	MS	F	p
3GRP	2	0.071	0.036	0.28	0.759
Error	100	9.648	0.129		
Total	101	9.719			

Level	N	Mean	StDev
1	17	0.9708	0.2031
2	12	0.8655	0.2141
3	73	0.8987	0.4022

Pooled StDev = 0.3587

Individual 95% CIs For Mean Based on Pooled StDev

```
-------+---------+---------+---------
       (-------------*------------)
(--------------*-------------)
          (------*-----)
-------+---------+---------+---------
     0.75      0.90      1.05
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

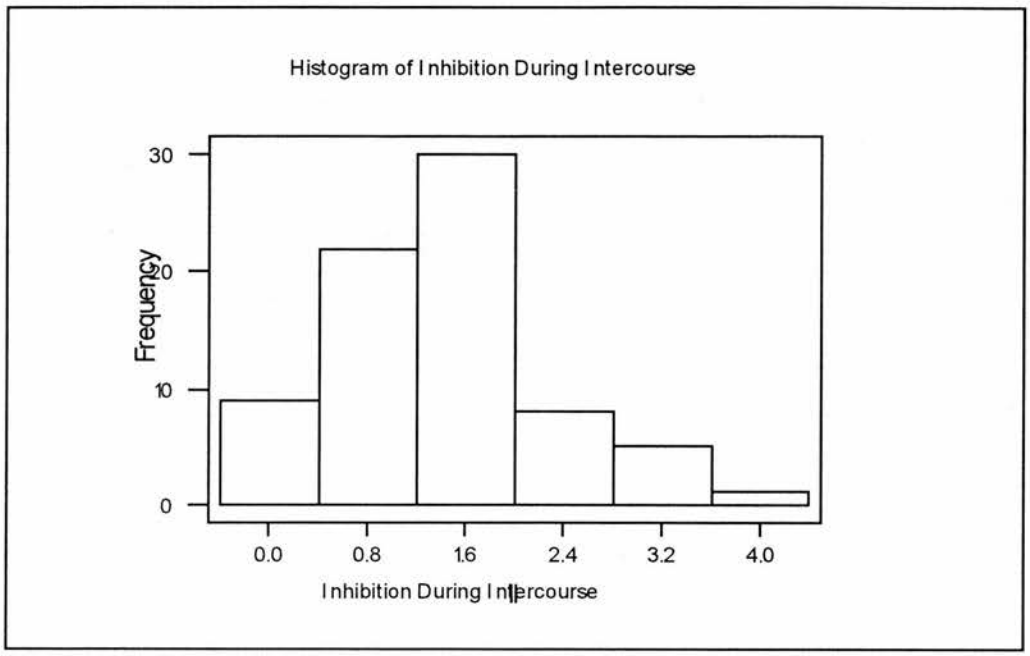
Critical value = 1.992

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.1928 0.4036	
3	-0.1555 0.2997	-0.2692 0.2027

MEN

Figure 17:ANOVA OF II (INHIBITION DURING INTERCOURSE) SUBSCALE OF SES 3



Analysis of Variance on II

Source	DF	SS	MS	F	p
GRP	2	3.537	1.768	2.31	0.107
Error	100	55.129	0.766		
Total	102	58.666			

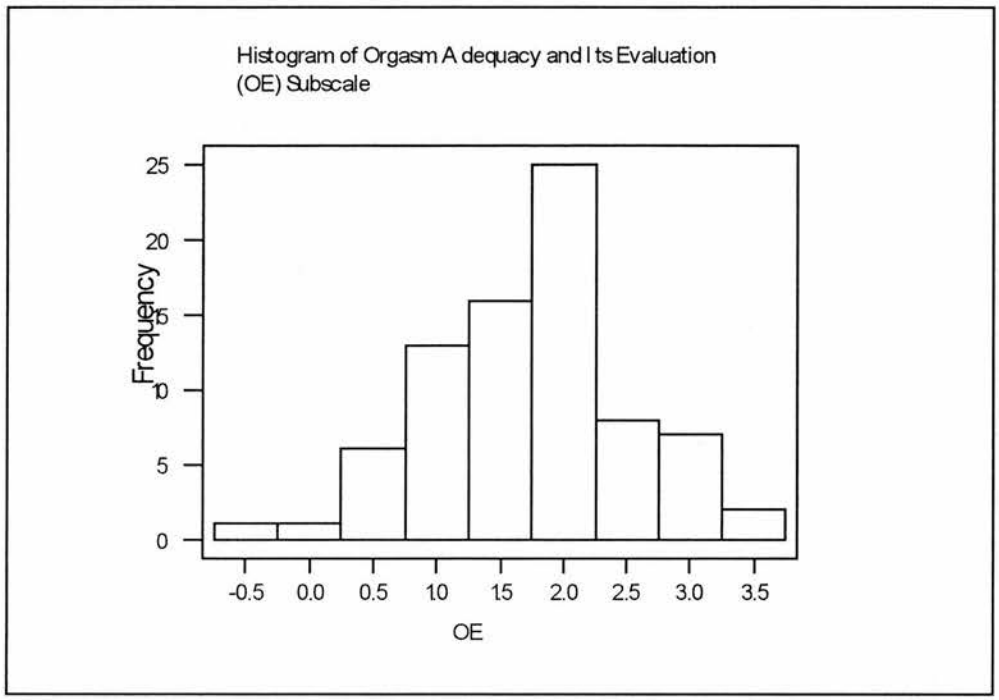
Level	N	Mean	StDev
1	17	1.3183	0.8065
2	12	0.9173	0.4877
3	73	1.5313	0.9455

Pooled StDev = 0.8750

Individual 95% CIs For Mean Based on Pooled StDev

MEN

Figure 18:ANOVA OF OE (ORGASM ADEQUACY DURING INTERCOURSE) SUBSCALE OF SES 3



Variable	N	Mean	Median	TrMean	StDev	SEMean
oe	102	1.7301	1.8600	1.7376	0.8003	0.0900

Variable	Min	Max	Q1	Q3
oe	-0.5300	3.5400	1.1200	2.1500

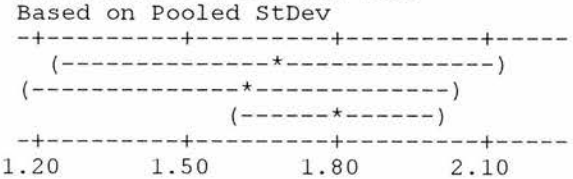
Analysis of Variance on OE

Source	DF	SS	MS	F	p
GRP	2	0.452	0.226	0.38	0.683
Error	100	44.333	0.591		
Total	101	44.785			

Level	N	Mean	StDev
1	17	1.6800	0.7347
2	12	1.6125	0.9125
3	73	1.8069	0.7426

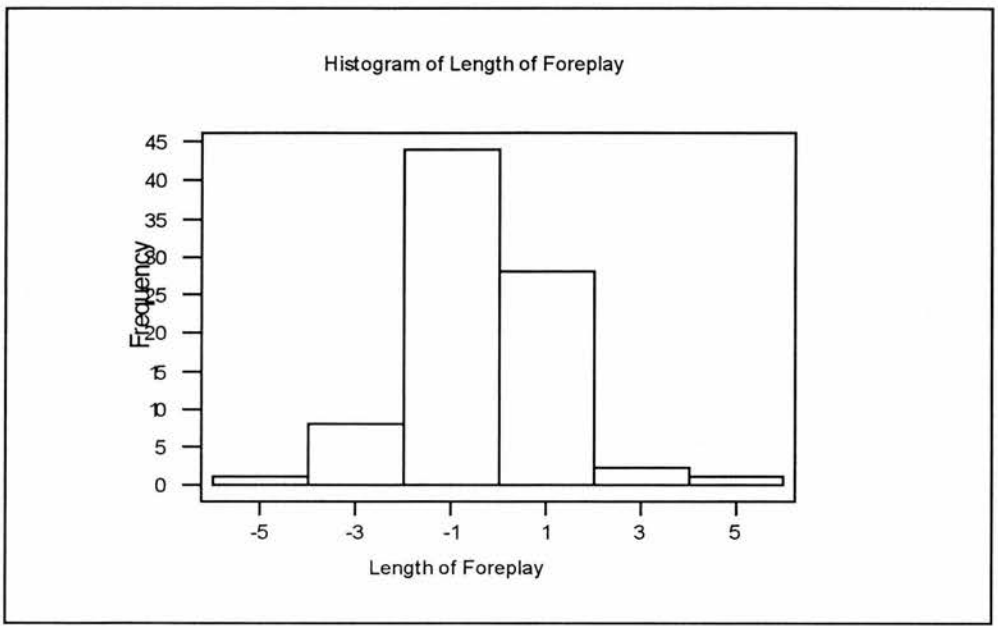
Pooled StDev = 0.7688

Individual 95% CIs For Mean Based on Pooled StDev



MEN

Figure 19:ANOVA OF LF (LENGTH OF FOREPLAY) SUBSCALE OF SES 3



Analysis of Variance on LF

Source	DF	SS	MS	F	p
GRP	2	2.15	1.07	0.50	0.608
Error	101	173.94	2.15		
Total	102	176.08			

Level	N	Mean	StDev
1	17	-0.259	1.232
2	12	-0.727	1.437
3	73	-0.274	1.524

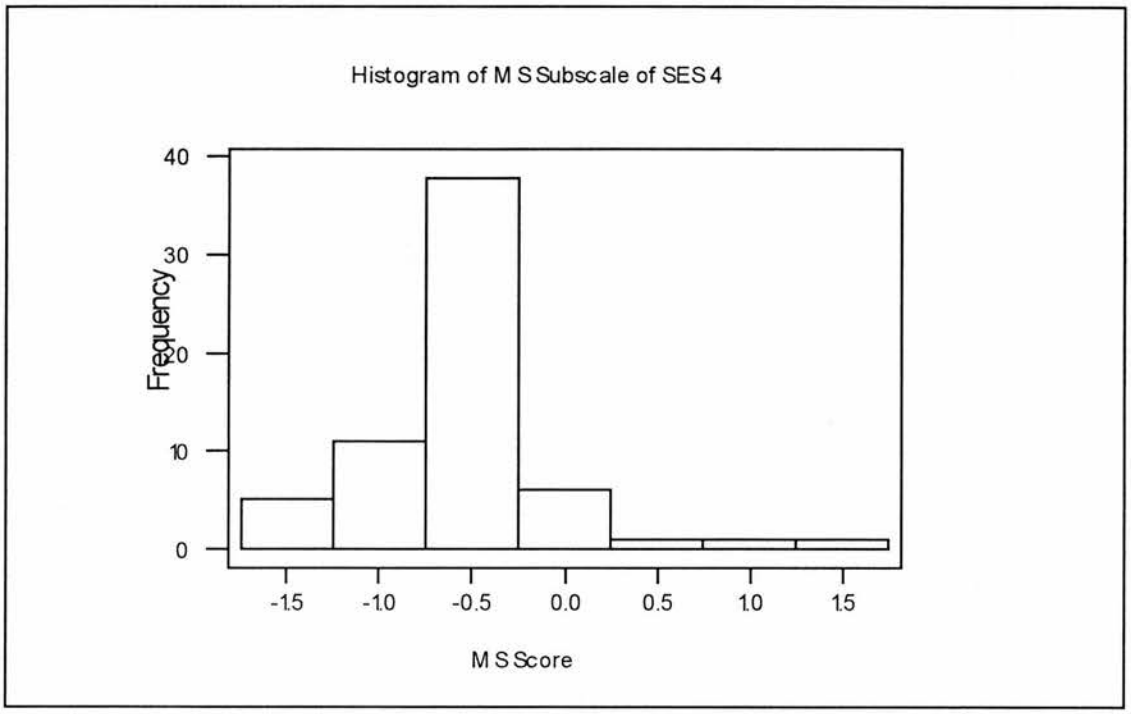
Pooled StDev = 1.465

Individual 95% CIs For Mean Based on Pooled StDev

```
-------+---------+---------+---------
          (------------*-----------)
 (-------------*-------------)
                  (-----*------)
-------+---------+---------+---------
    -1.20     -0.60      0.00
```

MEN

Figure 20:ANOVA OF MARITAL SATISFACTION SUBSCALE OF SES 4



Analysis of Variance on MS

Source	DF	SS	MS	F	p
GRP	2	1.273	0.636	3.21	0.046
Error	100	16.272	0.198		
Total	101	17.544			

Level	N	Mean	StDev
1	17	-0.3406	0.5630
2	12	-0.7183	0.4112
3	73	-0.6277	0.4153

Pooled StDev = 0.4455

Individual 95% CIs For Mean
Based on Pooled StDev

```
---------+---------+---------+-------
                   (-------*--------)
(---------*----------)
          (----*----)
---------+---------+---------+-------
      -0.75     -0.50     -0.25
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

Critical value = 1.989

Intervals for (column level mean) - (row level mean)

	1	2
2	0.0394 0.7161	
3	0.0364 0.5378	-0.3720 0.1908

MEN

Because distribution skewed result checked using non parametric test.

Kruskal-Wallis Test

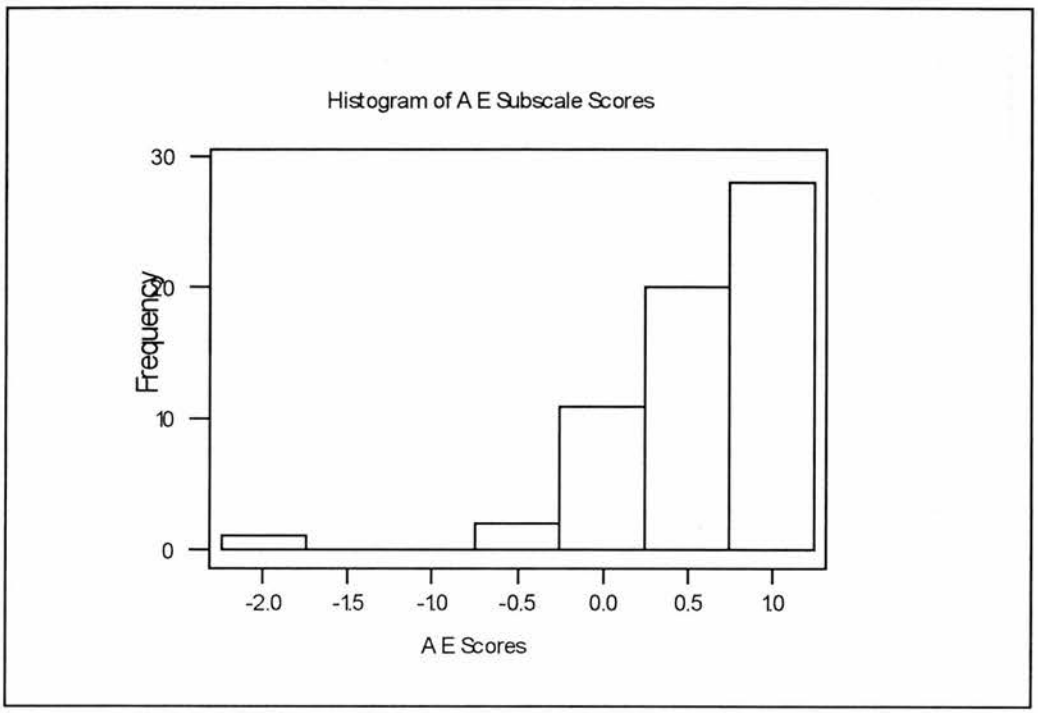
102 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	17	-0.4950	53.7	1.93
2	12	-0.5700	40.1	-0.44
3	73	-0.5900	40.6	-1.28
OVERALL	102		43.0	

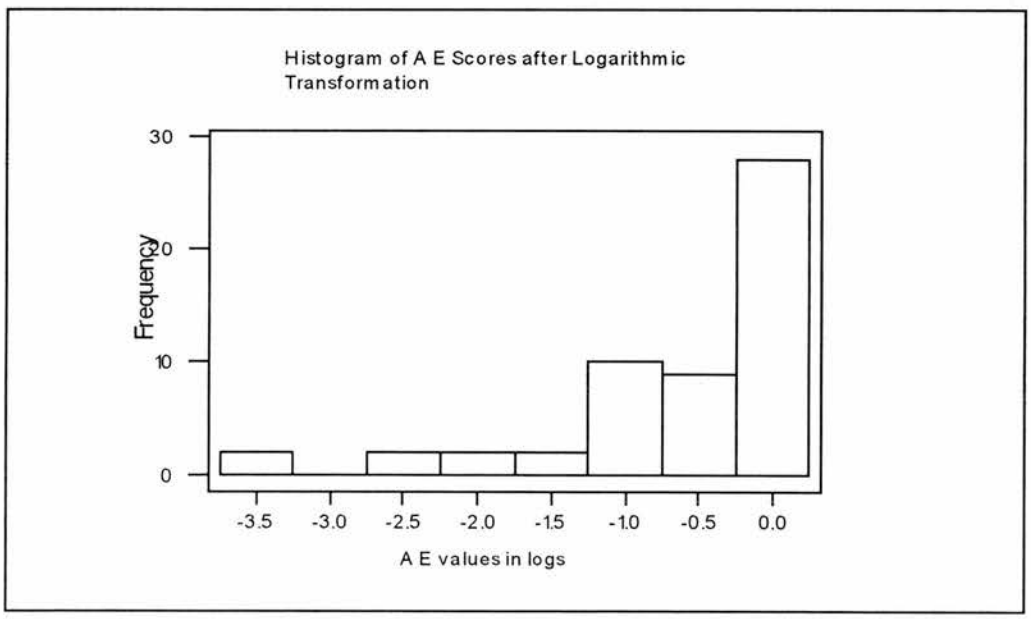
H = 3.72 d.f. = 2 p = 0.156
H = 3.73 d.f. = 2 p = 0.155 (adjusted for ties)

MEN

Figure 21:KRUSKALL-WALLIS OF AE (ATTITUDE TO EXTRAMARITAL INVOLVEMENT) OF SES 4



Because distribution is skew the values were transformed to logs



MEN

Because the distribution of values is skewed a the kruskall-wallis was employed.

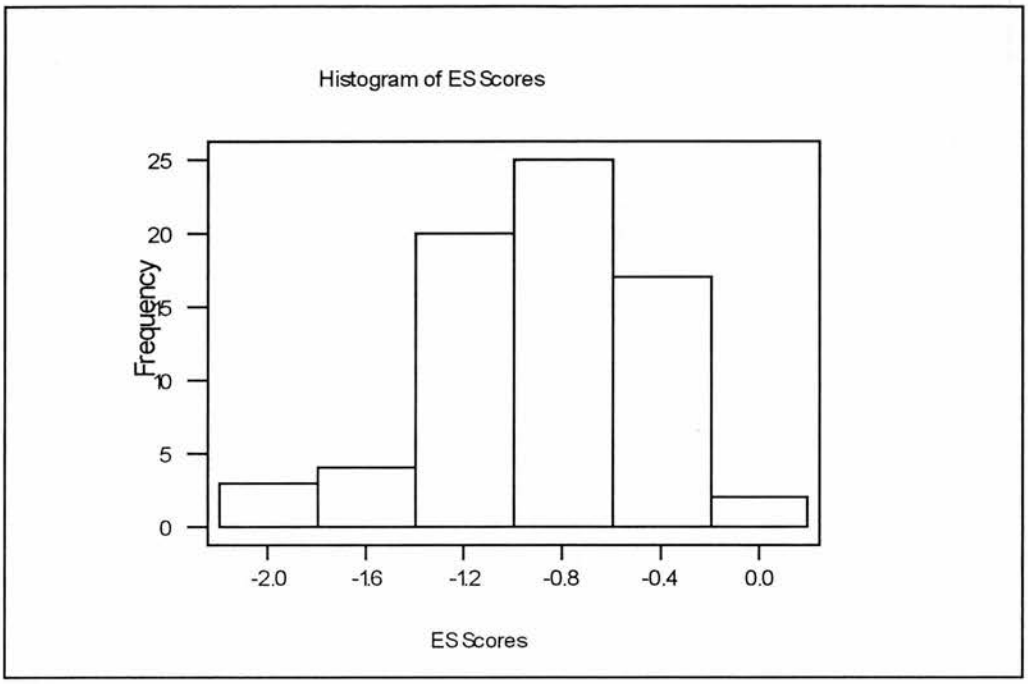
102 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	17	0.9700	44.3	2.59
2	12	0.6500	29.6	-0.34
3	72	0.5800	28.6	-1.86
OVERALL	102		31.5	

H = 6.73 d.f. = 2 p = 0.035
H = 6.77 d.f. = 2 p = 0.034 (adjusted for ties)

MEN

Figure 22:ANOVA OF ES (EVALUATION OF PARTNER AS SEXUAL PARTNER) SUBSCALE SUBSCALE OF SES 4



Analysis of Variance on ES

Source	DF	SS	MS	F	p
GRP	2	0.059	0.030	0.17	0.844
Error	100	11.913	0.175		
Total	101	11.972			

Level	N	Mean	StDev
1	17	-0.9192	0.2635
2	12	-0.8518	0.2140
3	73	-0.9333	0.4769

Pooled StDev = 0.4186

Individual 95% CIs For Mean
Based on Pooled StDev

```
---+---------+---------+---------+---
(--------------*--------------)
    (---------------*---------------)
       (-------*------)
---+---------+---------+---------+---
-1.12     -0.96     -0.80     -0.64
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

Critical value = 1.995

MEN

Figure 23:ANOVA OF MS (MORALITY WITHIN MARRIAGE) SUBSCALE OF SES 1 FOR SEIZURE TYPE GROUPS

```
Analysis of Variance on MS
Source      DF        SS        MS         F        p
SEIZTYP      3     4.048     1.349      5.44    0.001
Error      230    42.394     0.248
Total      231    46.443

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  -------+---------+---------+---------
 CPS        75    0.2304    0.5750                          (------*-----)
 PGE        86    0.1072    0.4492                    (-----*-----)
 UND        26    0.0994    0.6228              (-----------*-----------)
 CONT       45   -0.1793    0.4074  (-------*-------)
                                      -------+---------+---------+---------
Pooled StDev =    0.4979              -0.20      0.00      0.20

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.0597
          0.3060

     3   -0.1500   -0.2664
          0.4120    0.2822

     4    0.2058    0.0919   -0.0101
          0.6136    0.4811    0.5674


CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL
```

MEN

Figure 24:ANOVA OF PS (PREMARITAL SEXUAL EXPERIENCE) SUBSCALE OF SES 1 FOR SEIZURE TYPE GROUPS

```
Analysis of Variance on PS

Source      DF         SS         MS         F         p
SEIZTYP      3      0.076      0.025      0.03     0.993
Error      230    150.160      0.889
Total      231    150.236

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level       N       Mean      StDev  ----------+---------+---------+------
CPS         75    -0.4157     0.8396         (--------*--------)
PGE         86    -0.4595     1.0116         (-------*------)
UND         26    -0.4269     0.7711  (---------------*--------------)
CONT        45    -0.4640     0.9986       (---------*--------)
                                        ----------+---------+---------+------
Pooled StDev =    0.9426                       -0.60     -0.30      0.00

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

               1         2         3

     2    -0.3070
           0.3947

     3    -0.5246   -0.5512
           0.5469    0.4858

     4    -0.3429   -0.3628   -0.5095
           0.4396    0.3718    0.5838


CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL
```

MEN

Figure 25: ANOVA OF PA (PREMARITAL SEXUAL ATTITUDE) SUBSCALE OF SES 1 FOR SEIZURE TYPE GROUPS

```
Analysis of Variance on PA
Source     DF        SS        MS        F        p
SEIZTYP     3     0.266     0.089     0.59    0.620
Error     230    25.016     0.150
Total     231    25.283

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level      N      Mean     StDev  ---+---------+---------+---------+---
  CPS      75   -0.0726    0.4290   (------*------)
  PGE      86   -0.0274    0.3423       (-----*-----)
  UND      26    0.0780    0.5222       (------------*------------)
  CONT     45   -0.0388    0.3422     (-------*-------)
                                     ---+---------+---------+---------+---
Pooled StDev =    0.3870            -0.15      0.00      0.15      0.30

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.1889
          0.0985

     3   -0.3755   -0.3242
          0.0743    0.1135

     4   -0.1948   -0.1410   -0.1138
          0.1272    0.1638    0.3473


CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL
```

MEN

Figure 26:ANOVA OF AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE OF SES 2 FOR SEIZURE TYPE GROUPS

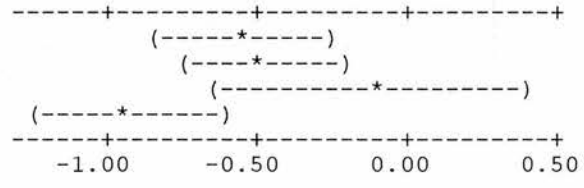
Analysis of Variance on AE

Source	DF	SS	MS	F	p
SEIZTYP	3	9.25	3.08	2.65	0.050
Error	230	197.72	1.16		
Total	231	206.97			

Level	N	Mean	StDev
CPS	75	-0.565	1.094
PGE	86	-0.477	1.127
UND	26	-0.112	1.087
CONT	45	-0.927	0.965

Pooled StDev = 1.078

Individual 95% CIs For Mean Based on Pooled StDev



Fisher's pairwise comparisons

Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.487 0.311		
3	-1.052 0.146	-0.942 0.212	
4	-0.092 0.815	0.026 0.874	0.199 1.431

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 27:ANOVA OF EE (EVALUATION OF EXPOSURE TO EROTIC IMAGERY) SUBSCALE OF SES 2 FOR SEIZURE TYPE GROUPS

```
Analysis of Variance on EE
Source     DF        SS        MS        F        p
SEIZTYP     3     0.551     0.184     0.31    0.815
Error     173   101.195     0.585
Total     176   101.746

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level      N      Mean     StDev  ------+---------+---------+---------+
 CPS       75   -0.2539    0.6851         (--------*-------)
 PGE       86   -0.2524    0.8083           (------*------)
 UND       26   -0.2587    1.1054  (---------------*---------------)
 CONT      45   -0.3837    0.6226  (---------*--------)
                                   ------+---------+---------+---------+
Pooled StDev =   0.7648               -0.50     -0.25      0.00      0.25

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.2821
          0.2790

     3   -0.4278   -0.4137
          0.4374    0.4265

     4   -0.1828   -0.1637   -0.3171
          0.4424    0.4263    0.5671
```

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 28:ANOVA OF IS (INTERPERSONAL SEXUAL ATTRACTION) SUBSCALE OF SES 2 FOR SEIZURE TYPE GROUPS

Analysis of Variance on IS

Source	DF	SS	MS	F	p
SEIZTYP	3	3.262	1.087	2.15	0.096
Error	230	86.497	0.506		
Total	231	89.759			

Level	N	Mean	StDev	Individual 95% CIs For Mean Based on Pooled StDev
CPS	75	-0.4616	0.6720	(-------*------)
PGE	86	-0.5785	0.7983	(------*------)
UND	26	-0.6340	0.8038	(--------------*-------------)
CONT	45	-0.8317	0.5590	(--------*--------)

Pooled StDev = 0.7112

-1.00 -0.75 -0.50 -0.25

Fisher's pairwise comparisons

Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.1440 0.3778		
3	-0.2399 0.5848	-0.3455 0.4565	
4	0.0776 0.6626	-0.0232 0.5295	-0.2246 0.6200

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 29:ANOVA OF FI (PREFERRED FREQUENCY OF INTERCOURSE) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

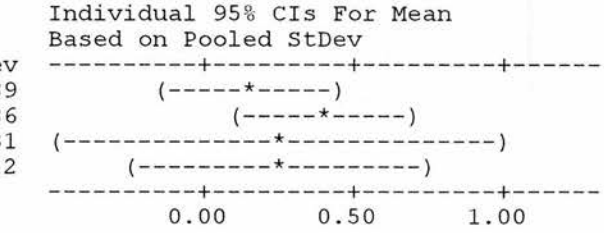
Analysis of Variance on FI

Source	DF	SS	MS	F	p
SEIZTYP	3	0.800	0.267	0.39	0.763
Error	100	49.649	0.690		
Total	101	50.449			

Level	N	Mean	StDev
CPS	38	0.1638	0.8689
PGE	36	0.3928	0.7786
UND	11	0.2480	1.0231
CONT	17	0.2380	0.7642

Pooled StDev = 0.8304

Individual 95% CIs For Mean Based on Pooled StDev



Fisher's pairwise comparisons

Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.993

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.6566 0.1985		
3	-0.8818 0.7134	-0.6566 0.9462	
4	-0.6550 0.5066	-0.4312 0.7409	-0.8826 0.9026

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 30:ANOVA OF EP (ENJOYMENT POTENTIAL OF INTERCOURSE) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

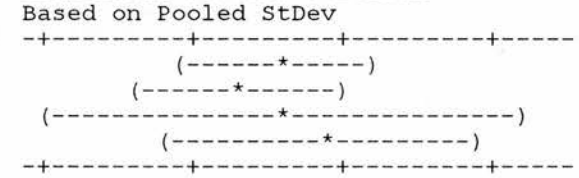
Analysis of Variance on EP

Source	DF	SS	MS	F	p
SEIZTYP	3	0.124	0.041	0.32	0.813
Error	100	9.344	0.130		
Total	101	9.468			

Level	N	Mean	StDev
CPS	38	0.9110	0.4334
PGE	36	0.8546	0.3315
UND	11	0.9200	0.2687
CONT	17	0.9708	0.2031

Pooled StDev = 0.3602

Individual 95% CIs For Mean Based on Pooled StDev



Fisher's pairwise comparisons

Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.993

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.1309 0.2435		
3	-0.3550 0.3370	-0.4139 0.2832	
4	-0.3040 0.1842	-0.3639 0.1315	-0.4330 0.3313

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 31:ANOVA OF LF (LENGTH OF FOREPLAY) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on LF

```
Source      DF        SS        MS        F        p
SEIZTYP      3      0.41      0.14     0.06    0.978
Error      100    175.05      2.11
Total      101    175.46

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  ----------+---------+---------+------
 CPS        38    -0.294     1.158            (-----*------)
 PGE        36    -0.418     1.815       (------*-----)
 UND        11    -0.155     1.487  (-- -------------*-----------------)
 CONT       17    -0.323     1.175         (--------*--------)
                                      ----------+---------+---------+------
Pooled StDev =     1.452                      -0.80      0.00      0.80

Fisher's pairwise comparisons

    Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.989

Intervals for (column level mean) - (row level mean)

               1         2         3

     2    -0.587
           0.836

     3    -1.665    -1.795
           1.388     1.269

     4    -0.829    -0.962    -1.437
           0.887     0.772     1.773
```

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 32:ANOVA OF OE (ORGASM ADEQUACY DURING INTERCOURSE AND ITS EVALUATION) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on OE

```
Source      DF        SS        MS        F        p
SEIZTYP      3     0.340     0.113     0.19    0.906
Error       71    43.296     0.610
Total       74    43.636

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  -----+---------+---------+---------+-
 CPS        38    1.8223    0.8285               (------*------)
 PGE        36    1.6850    0.7544          (------*------)
 UND        11    1.7040    0.6967      (------------*-------------)
 CONT       17    1.6800    0.7347      (-----------*-----------)
                                      -----+---------+---------+---------+-
Pooled StDev =    0.7809               1.20      1.60      2.00      2.40

Fisher's pairwise comparisons

    Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.994

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.2687
          0.5432

     3   -0.6322   -0.7750
          0.8687    0.7370

     4   -0.4042   -0.5491   -0.8158
          0.6887    0.5591    0.8638


CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL
```

MEN

Figure 33: ANOVA OF II (INHIBITION DURING INTERCOURSE) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on II

```
Source      DF        SS        MS         F        p
SEIZTYP      3     1.127     0.376      0.49    0.694
Error       66    51.074     0.774
Total       69    52.200

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  -------+---------+---------+---------
 CPS        38    1.5950    0.8981              (------*------)
 PGE        36    1.3535    0.9028         (------*------)
 UND        11    1.4440    0.8965       (----------*-----------)
 CONT       17    1.2862    0.7808     (---------*--------)
                                      -------+---------+---------+---------
Pooled StDev =    0.8797                 1.00      1.50      2.00

Fisher's pairwise comparisons

    Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.997

Intervals for (column level mean) - (row level mean)

               1         2         3

     2    -0.2457
           0.7288

     3    -0.7069   -0.9484
           1.0089    0.7673

     4    -0.2879   -0.5294   -0.7666
           0.9056    0.6640    1.0823


CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL
```

MEN

Figure 34: ANOVA OF MS (MARITAL SATISFACTION) SUBSCALE OF SES 4 FOR SEIZURE TYPE GROUPS

Analysis of Variance on MS

Source	DF	SS	MS	F	p
SEIZTYP	3	1.332	0.444	2.23	0.092
Error	100	14.966	0.200		
Total	101	16.298			

Level	N	Mean	StDev	Individual 95% CIs For Mean Based on Pooled StDev
CPS	38	-0.6835	0.5271	(------*-----)
PGE	36	-0.6375	0.2622	(------*-----)
UND	11	-0.5625	0.0838	(----------*----------)
CONT	17	-0.3406	0.5630	(--------*--------)

Pooled StDev = 0.4467 -1.00 -0.75 -0.50 -0.25

Fisher's pairwise comparisons

Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.992

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.2780 0.1859		
3	-0.5938 0.3517	-0.5506 0.4006	
4	-0.6168 -0.0690	-0.5757 -0.0180	-0.7193 0.2756

Kruskal-Wallis Test

102 CASES

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
CPS	36	-0.6300	35.6	-1.37
PGE	32	-0.5400	38.5	-0.43
UND	7	-0.5900	41.4	0.12
CONT	27	-0.4950	50.8	2.10
OVERALL	79		40.0	

H = 4.77 d.f. = 3 p = 0.190
H = 4.79 d.f. = 3 p = 0.189 (adjusted for ties)

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 35:ANOVA OF ES (EVALUATION OF PARTNER AS SEXUAL PARTNER) SUBSCALE OF SES 4 FOR SEIZURE TYPE GROUPS

Analysis of Variance on ES

Source	DF	SS	MS	F	p
SEIZTYP	3	0.286	0.095	0.58	0.630
Error	100	10.515	0.164		
Total	102	10.801			

Level	N	Mean	StDev	Individual 95% CIs For Mean Based on Pooled StDev
CPS	36	-1.0152	0.3982	(-----*------)
PGE	32	-0.8780	0.4595	(------*-----)
UND	7	-0.8500	0.4322	(---------------*---------------)
CONT	27	-0.9192	0.2635	(--------*---------)

Pooled StDev = 0.4053

-1.25 -1.00 -0.75 -0.50

Fisher's pairwise comparisons

Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.998

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.3620 0.0876		
3	-0.5991 0.2687	-0.4641 0.4081	
4	-0.3770 0.1850	-0.2432 0.3256	-0.3984 0.5367

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 36:KRUSKALL-WALLIS AE (ATTITUDE TO EXTRAMARITAL INVOLVEMENT) SUBSCALE OF SES 4 FOR SEIZURE TYPE GROUPS

102 CASES

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
CPS	36	-0.9700	30.9	-1.22
PGE	32	-0.8800	37.9	1.09
UND	7	-0.8900	38.3	0.39
CONT	27	-0.9200	34.2	-0.06
OVERALL	68		34.5	

H = 1.80 d.f. = 3 p = 0.614
H = 1.81 d.f. = 3 p = 0.614 (adjusted for ties)

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERALISED EPILEPSY
UND = UNDEFINED
CONT = CONTROL

MEN

Figure 37 :MULTIPLE REGRESSION FOR SES 1 (UNWEIGHTED) SCORES

The regression equation is
SES1 = 53.1 + 0.219 AGE + 3.00 AFFIL - 10.8 FURTHED - 1.57 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	53.098	3.461	15.34	0.000
AGE	0.2194	0.1143	1.92	0.056
AFFIL	2.996	1.737	1.72	0.086
FURTHED	-10.800	1.737	-6.22	0.000
MARRIED?	-1.574	2.009	-0.78	0.434

s = 12.58 R-sq = 18.4% R-sq(adj) = 16.9%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	7363.6	1840.9	11.64	0.000
Error	230	32576.9	158.1		
Total	231	39940.5			

SOURCE	DF	SEQ SS
AGE	1	513.2
AFFIL	1	661.5
FURTHED	1	6091.9
MARRIED?	1	97.0

Stepwise Regression

F-to-Enter: 4.00 F-to-Remove: 4.00

Response is SES1 on 3 predictors, with N = 232

Step	1
Constant	60.77
FURTHED	-10.8
T-Ratio	-6.26
S	12.9
R-Sq	14.94

MEN

Figure 38:MULTIPLE REGRESSION FOR SES 2 (UNWEIGHTED) SCORES

The regression equation is
SES2 = 39.9 + 0.0191 AGE + 0.97 AFFIL - 4.13 FURTHED + 0.65 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	39.936	2.493	16.02	0.000
AGE	0.01912	0.08233	0.23	0.817
AFFIL	0.973	1.251	0.78	0.438
FURTHED	-4.130	1.251	-3.30	0.001
MARRIED?	0.648	1.447	0.45	0.655

s = 9.058 R-sq = 5.7% R-sq(adj) = 3.8%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	1014.66	253.66	3.09	0.017
Error	230	16902.77	82.05		
Total	231	17917.43			

SOURCE	DF	SEQ SS
AGE	1	23.58
AFFIL	1	75.93
FURTHED	1	898.70
MARRIED?	1	16.45

Stepwise Regression

F-to-Enter: 4.00 F-to-Remove: 4.00

Response is SES2 on 3 predictors, with N = 232

Step	1
Constant	41.54
FURTHED	-4.4
T-Ratio	-3.67
S	8.98
R-Sq	5.69

MEN

Figure 39:MULTIPLE REGRESSION FOR SES 3 (UNWEIGHTED) SCORES

The regression equation is
SES3 = 41.6 - 5.88 FURTHED - 3.20 AFFIL + 0.397 AGE

101 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	41.601	7.555	5.51	0.000
FURTHED	-5.878	3.363	-1.75	0.084
AFFIL	-3.198	3.381	-0.95	0.347
AGE	0.3965	0.1991	1.99	0.050

s = 15.95 R-sq = 8.1% R-sq(adj) = 4.8%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	1915.6	638.5	2.51	0.064
Error	99	21872.9	254.3		
Total	100	23788.5			

SOURCE	DF	SEQ SS
FURTHED	1	759.7
AFFIL	1	147.0
AGE	1	1008.9

Stepwise Regression

F-to-Enter: 4.00 F-to-Remove: 4.00

Response is SES3 on 2 predictors, with N = 101

Step	1
Constant	32.10
AGE	0.51
T-Ratio	2.67
S	16.0
R-Sq	6.86

MEN

Figure 40: MULTIPLE REGRESSION FOR SES 4 (UNWEIGHTED) SCORES

The regression equation is
SES 4 = 63.8 + 0.27 FURTHER EDUCATION + 0.089 AGE + 2.03 AFFIL

```
101 cases
Predictor       Coef       Stdev    t-ratio        p
Constant      63.797       4.871      13.10    0.000
FURTHED        0.270       2.182       0.12    0.902
AGE           0.0887      0.1304       0.68    0.498
AFFIL          2.032       2.184       0.93    0.355

s = 10.54      R-sq = 1.6%     R-sq(adj) = 0.0%

Analysis of Variance

SOURCE        DF          SS          MS         F        p
Regression     3       164.0        54.7      0.49    0.689
Error         99      9998.0       111.1
Total        100     10161.9

SOURCE        DF      SEQ SS
FURTHED        1         3.0
AGE            1        64.8
AFFIL          1        96.1
```

Figure 41:MULTIPLE REGRESSION FOR PA (PREMARITAL SEXUAL ATTITUDE) SUBSCALE OF SES 1

The regression equation is

PA = - 0.230 + 0.00527 AGE - 0.0301 AFFIL + 0.112 FURTHED - 0.0292 MARRIED?

```
232 cases
Predictor       Coef       Stdev    t-ratio        p
Constant     -0.2303      0.1182      -1.95    0.053
AGE         0.005267    0.003982       1.32    0.188
AFFIL       -0.03010     0.06170      -0.49    0.626
FURTHED      0.11221     0.06155       1.82    0.070
MARRIED?    -0.02917     0.07049      -0.41    0.680

s = 0.4041      R-sq = 3.3%      R-sq(adj) = 1.0%

Analysis of Variance

SOURCE        DF          SS          MS         F        p
Regression     4      0.9331      0.2333      1.43    0.227
Error        231     27.5983      0.1633
Total        232     28.5314

SOURCE        DF      SEQ SS
AGE            1      0.2945
AFFIL          1      0.0587
FURTHED        1      0.5519
MARRIED?       1      0.0280
```

MEN

Figure 42: MULTIPLE REGRESSION FOR MS (SEXUAL MORALITY WITHIN MARRIAGE) SUBSCALE OF SES 1

The regression equation is

MS = - 0.001 + 0.00850 AGE + 0.0196 AFFIL - 0.347 FURTHED - 0.0888 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.0012	0.1425	-0.01	0.993
AGE	0.008502	0.004834	1.76	0.080
AFFIL	0.01957	0.07422	0.26	0.792
FURTHED	-0.34670	0.07419	-4.67	0.000
MARRIED?	-0.08883	0.08632	-1.03	0.305

s = 0.4919 R-sq = 12.1% R-sq(adj) = 10.1%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	5.7886	1.4471	5.98	0.000
Error	230	41.8590	0.2420		
Total	231	47.6476			

SOURCE	DF	SEQ SS
AGE	1	0.4070
AFFIL	1	0.0128
FURTHED	1	5.1126
MARRIED?	1	0.2562

Stepwise Regression

F-to-Enter: 4.00 F-to-Remove: 4.00

Response is MS on 2 predictors, with N = 232

Step	1
Constant	0.2253
FURTHED	-0.327
T-Ratio	-4.60
S	0.492
R-Sq	10.02

MEN

Figure 43: MULTIPLE REGRESSION FOR AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE OF SES 2

The regression equation is

AE = - 0.012 - 0.0102 AGE - 0.025 AFFIL - 0.529 FURTHED + 0.136 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.0123	0.3044	-0.04	0.968
AGE	-0.01023	0.01010	-1.01	0.313
AFFIL	-0.0245	0.1551	-0.16	0.875
FURTHED	-0.5293	0.1550	-3.41	0.001
MARRIED?	0.1361	0.1763	0.77	0.441

s = 1.053 R-sq = 6.8% R-sq(adj) = 4.8%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	14.825	3.706	3.34	0.011
Error	230	201.788	1.109		
Total	231	216.613			

SOURCE	DF	SEQ SS
AGE	1	0.791
AFFIL	1	0.000
FURTHED	1	13.373
MARRIED?	1	0.661

Stepwise Regression

F-to-Enter: 4.00 F-to-Remove: 4.00

Response is AE on 3 predictors, with N = 232

Step	1
Constant	-0.2769
FURTHED	-0.54
T-Ratio	-3.51
S	1.05
R-Sq	6.24

MEN

Figure 44: MULTIPLE REGRESSION FOR AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE OF SES 2

The regression equation is

AE = - 0.012 - 0.0102 AGE - 0.025 AFFIL - 0.529 FURTHED + 0.136 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.0123	0.3044	-0.04	0.968
AGE	-0.01023	0.01010	-1.01	0.313
AFFIL	-0.0245	0.1551	-0.16	0.875
FURTHED	-0.5293	0.1550	-3.41	0.001
MARRIED?	0.1361	0.1763	0.77	0.441

s = 1.053 R-sq = 6.8% R-sq(adj) = 4.8%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	14.825	3.706	3.34	0.011
Error	230	201.788	1.109		
Total	231	216.613			

SOURCE	DF	SEQ SS
AGE	1	0.791
AFFIL	1	0.000
FURTHED	1	13.373
MARRIED?	1	0.661

Figure 45: MULTIPLE REGRESSION FOR EE (EVALUATION OF EXPOSURE TO EROTIC IMAGERY) SUBSCALE OF SES 2

The regression equation is

EE = - 0.263 - 0.00218 AGE + 0.122 AFFIL + 0.016 FURTHER EDUCATION- 0.070 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.2628	0.2269	-1.16	0.248
AGE	-0.002175	0.007678	-0.28	0.777
AFFIL	0.1215	0.1170	1.04	0.300
FURTHED	0.0157	0.1166	0.13	0.893
MARRIED?	-0.0696	0.1342	-0.52	0.605

s = 0.7819 R-sq = 0.9% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	0.9728	0.2432	0.40	0.810
Error	230	107.6137	0.6114		
Total	231	108.5864			

SOURCE	DF	SEQ SS
AGE	1	0.1492
AFFIL	1	0.6418
FURTHED	1	0.0175
MARRIED?	1	0.1642

MEN

Figure 46:MULTIPLE REGRESSION FOR IS (INTERPERSONAL SEXUAL ATTRACTION) SUBSCALE OF SES 2

The regression equation is

IS = - 0.851 + 0.00433 AGE + 0.124 AFFIL - 0.134 FURTHER EDUCATION + 0.173 MARRIED?

232 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.8513	0.1966	-4.33	0.000
AGE	0.004330	0.006756	0.64	0.522
AFFIL	0.1235	0.1020	1.21	0.227
FURTHED	-0.1344	0.1015	-1.32	0.187
MARRIED?	0.1727	0.1171	1.47	0.142

s = 0.6744 R-sq = 4.7% R-sq(adj) = 2.5%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	3.8937	0.9734	2.14	0.078
Error	173	78.6835	0.4548		
Total	177	82.5772			

SOURCE	DF	SEQ SS
AGE	1	1.2528
AFFIL	1	0.7243
FURTHED	1	0.9272
MARRIED?	1	0.9894

Figure 47: MULTIPLE REGRESSION FOR FI (PREFERRED FREQUENCY OF INTERCOURSE) SUBSCALE OF SES3

The regression equation is

FI = 0.025 + 0.0108 AGE - 0.279 AFFIL + 0.081 FURTHER EDUCATION

102 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	0.0249	0.4146	0.06	0.952
AGE	0.01077	0.01106	0.97	0.334
AFFIL	-0.2795	0.1884	-1.48	0.142
FURTHED	0.0809	0.1910	0.42	0.673

s = 0.8116 R-sq = 4.4% R-sq(adj) = 0.4%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	2.1520	0.7173	1.09	0.359
Error	100	46.7651	0.6587		
Total	101	48.9171			

SOURCE	DF	SEQ SS
AGE	1	0.6121
AFFIL	1	1.4218
FURTHED	1	0.1181

MEN

Figure 48: MULTIPLE REGRESSION FOR EP (ENJOYMENT POTENTIAL OF INTERCOURSE) SUBSCALE OF SES3

The regression equation is

EP = 0.834 + 0.00471 AGE - 0.148 AFFILIATION - 0.0320 FURTHER EDUCATION

102 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	0.8340	0.1605	5.20	0.000
AGE	0.004713	0.004362	1.08	0.284
AFFIL	-0.14753	0.07321	-2.02	0.052
FURTHED	-0.03198	0.07361	-0.43	0.665

s = 0.3046 R-sq = 6.8% R-sq(adj) = 2.6%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	0.45220	0.15073	1.62	0.192
Error	100	6.21746	0.09280		
Total	101	6.66966			

SOURCE	DF	SEQ SS
AGE	1	0.05288
AFFIL	1	0.38180
FURTHED	1	0.01751

Figure 49:MULTIPLE REGRESSION FOR II (INHIBITION DURING INTERCOURSE) SUBSCALE OF SES3

The regression equation is

II = 0.659 + 0.0165 AGE + 0.220 AFFILIATION + 0.058 FURTHER EDUCATION

102 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	0.6590	0.4669	1.41	0.163
AGE	0.01652	0.01246	1.33	0.190
AFFIL	0.2203	0.2162	1.02	0.312
FURTHED	0.0578	0.2162	0.27	0.790

s = 0.8908 R-sq = 4.6% R-sq(adj) = 0.2%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	2.4666	0.8222	1.04	0.383
Error	65	51.5800	0.7935		
Total	68	54.0466			

SOURCE	DF	SEQ SS
AGE	1	1.6129
AFFIL	1	0.7969
FURTHED	1	0.0567

MEN

Figure 50:MULTIPLE REGRESSION LF (LENGTH OF FOREPLAY) SUBSCALE OF SES 3

The regression equation is

LF = - 1.55 + 0.0415 AGE - 0.334 AFFIL - 0.182 FURTHER EDUCATION

102 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-1.5491	0.7413	-2.09	0.040
AGE	0.04155	0.02016	2.06	0.053
AFFIL	-0.3336	0.3477	-0.96	0.341
FURTHED	-0.1815	0.3499	-0.52	0.605

s = 1.506 R-sq = 6.3% R-sq(adj) = 2.4%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	11.017	3.672	1.62	0.192
Error	72	163.255	2.267		
Total	75	174.272			

SOURCE	DF	SEQ SS
AGE	1	8.347
AFFIL	1	2.059
FURTHED	1	0.610

Figure 51: MULTIPLE REGRESSION OF OE (ORGASM ADEQUACY DURING INTERCOURSE) SUBSCALE OF SES 3

The regression equation is

OE = 1.51 + 0.0104 AGE - 0.067 AFFILIATION - 0.117 FURTHER EDUCATION

102 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	1.5083	0.3961	3.81	0.000
AGE	0.01038	0.01075	0.97	0.338
AFFIL	-0.0668	0.1818	-0.37	0.715
FURTHED	-0.1172	0.1832	-0.64	0.525

s = 0.7502 R-sq = 1.9% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	0.7076	0.2359	0.42	0.740
Error	66	37.1449	0.5628		
Total	69	37.8524			

SOURCE	DF	SEQ SS
AGE	1	0.3882
AFFIL	1	0.0889
FURTHED	1	0.2304

MEN

Figure 52:MULTIPLE REGRESSION OF MS (MARITAL SATISFACTION) SUBSCALE OF SES 4

The regression equation is

MS = - 0.472 - 0.00506 AGE + 0.024 AFFIL + 0.095 FURTHED

```
102 CASES
Predictor       Coef        Stdev    t-ratio        p
Constant     -0.4718       0.2339      -2.02    0.047
AGE       -0.005061     0.006339      -0.80    0.427
AFFIL        0.0237       0.1093       0.22    0.829
FURTHED      0.0952       0.1098       0.87    0.389

s = 0.4767      R-sq = 1.7%      R-sq(adj) = 0.0%

Analysis of Variance

SOURCE       DF          SS          MS         F        p
Regression    3      0.2835      0.0945      0.42    0.742
Error       100     16.5879      0.2272
Total       101     16.8715

SOURCE       DF      SEQ SS
AGE           1      0.1017
AFFIL         1      0.0110
FURTHED       1      0.1709
```

Figure 53: MULTIPLE REGRESSION OF ES (EVALUATION OF PARTNER AS SEXUAL PARTNER) SUBSCALE OF SES 4

The regression equation is
ES = - 0.812 - 0.00420 AGE + 0.056 AFFIL + 0.019 FURTHED

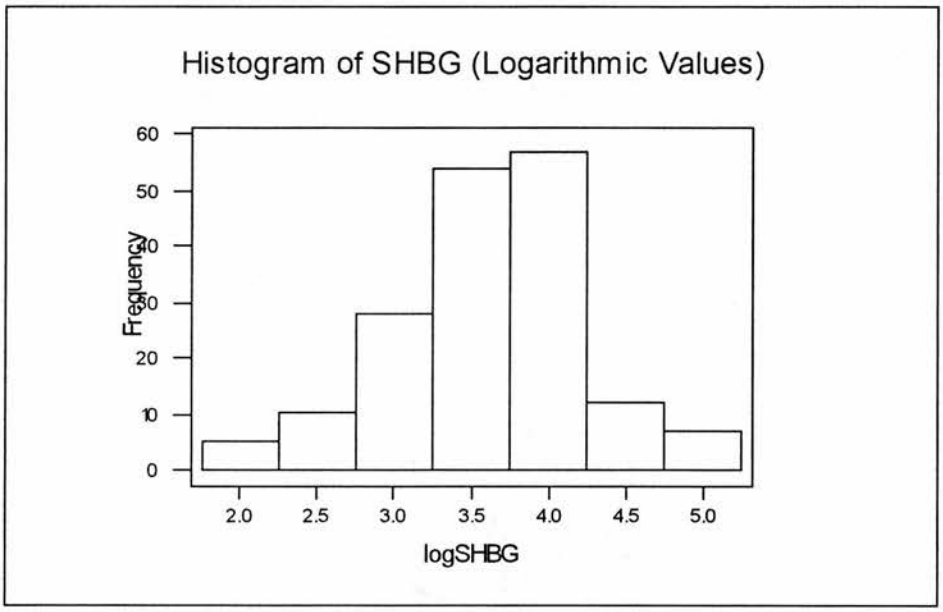
```
102 CASES

Predictor       Coef        Stdev    t-ratio        p
Constant     -0.8118       0.2415      -3.36    0.001
AGE       -0.004200     0.006449      -0.65    0.517
AFFIL        0.0560       0.1090       0.51    0.609
FURTHED      0.0191       0.1105       0.17    0.863

s = 0.4297      R-sq = 1.1%      R-sq(adj) = 0.0%

Analysis of Variance

SOURCE       DF          SS          MS         F        p
Regression    3      0.1191      0.0397      0.22    0.886
Error        59     10.8914      0.1846
Total        62     11.0105

SOURCE       DF      SEQ SS
AGE           1      0.0633
AFFIL         1      0.0502
FURTHED       1      0.0055
```

MEN

Figure 54: ANOVA OF SEX HORMONE BINDING GLOBULIN

(Values in μMol/L)

Because SHBG values not normally distributed they were transformed to logarithms



Analysis of Variance on logSHBG

Source	DF	SS	MS	F	p
GRP	5	15.453	3.091	10.82	0.000
Error	180	47.710	0.286		
Total	182	63.163			

Level	N	Mean	StDev
1	33	3.1514	0.5176
2	32	3.4049	0.4441
3	18	3.4183	0.6182
4	31	3.7496	0.5315
5	21	3.8605	0.6617
6	48	3.9424	0.5042

Pooled StDev = 0.5345

Individual 95% CIs For Mean
Based on Pooled StDev

```
------+---------+---------+---------+
 (----*----)
        (----*-----)
      (-------*------)
                (-----*-----)
                   (-----*------)
                       (----*---)
------+---------+---------+---------+
    3.15      3.50      3.85      4.20
```

MEN

Fisher's pairwise comparisons

Family error rate = 0.362
Individual error rate = 0.0500

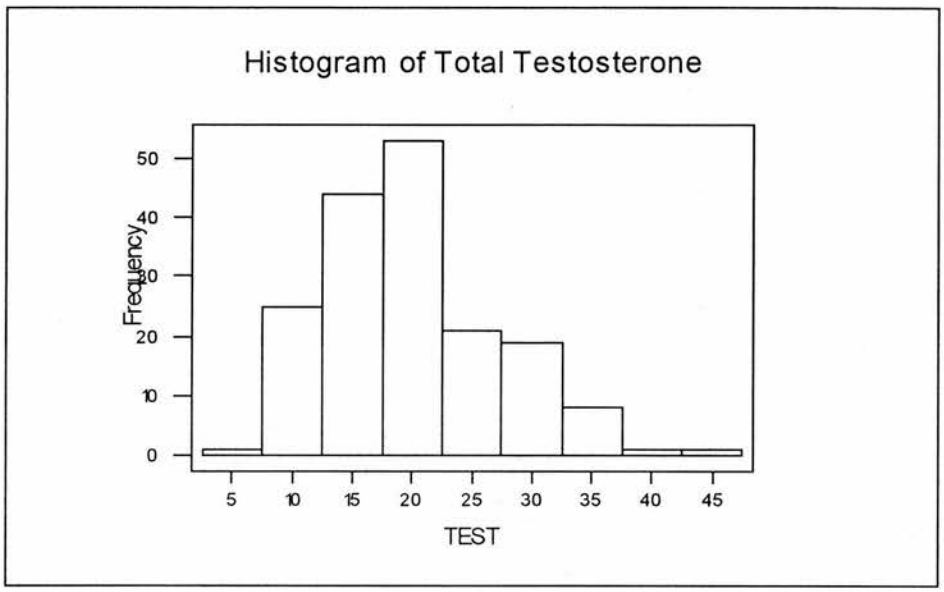
Critical value = 1.974

Intervals for (column level mean) - (row level mean)

	1	2	3	4	5
2	-0.5217 0.0146				
3	-0.5836 0.0497	-0.3336 0.3070			
4	-0.8713 -0.3252	-0.6219 -0.0674	-0.6558 -0.0069		
5	-1.0055 -0.4129	-0.7558 -0.1554	-0.7865 -0.0980	-0.4155 0.1937	
6	-1.0350 -0.5470	-0.7861 -0.2887	-0.8245 -0.2237	-0.4467 0.0612	-0.3607 0.1970

MEN

Figure 55: ANOVA TOTAL TESTOSTEONE (ηMol/L)



	N	Mean	Median	TrMean	StDev	SEMean
TESTOSTERONE	182	19.765	19.000	19.424	7.079	0.538

	Min	Max	Q1	Q3
TESTOSTERONE	7.400	44.000	14.000	23.700

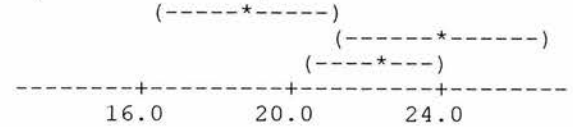
Analysis of Variance on TOTAL TESTOSTERONE

Source	DF	SS	MS	F	p
GRP	5	1229.5	245.9	5.56	0.000
Error	180	7390.0	44.3		
Total	182	8619.5			

Level	N	Mean	StDev
1	33	16.650	5.026
2	32	19.313	5.022
3	18	16.212	6.698
4	31	18.750	5.718
5	21	24.114	9.082
6	48	22.224	7.724

Pooled StDev = 6.652

Individual 95% CIs For Mean
Based on Pooled StDev

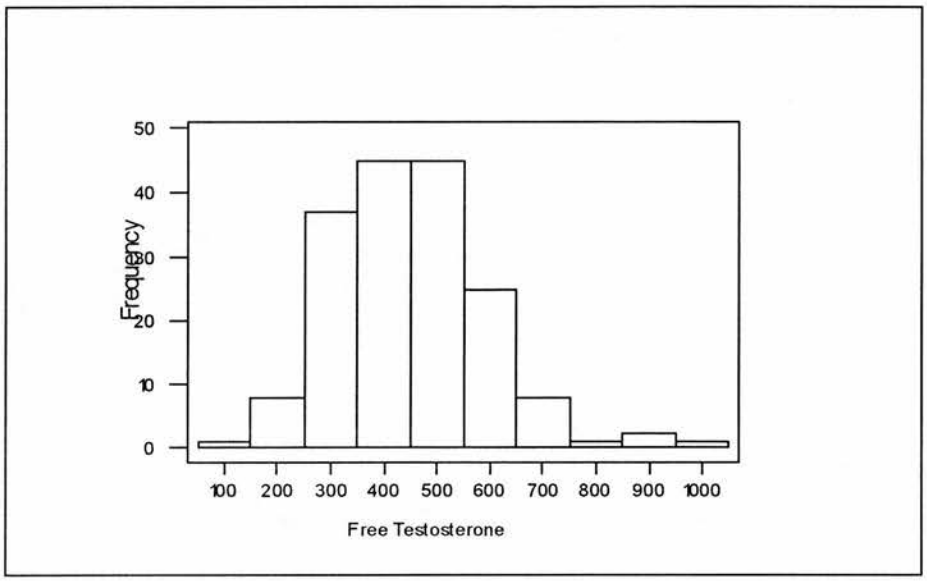


MEN

```
Fisher's pairwise comparisons

    Family error rate = 0.362
Individual error rate = 0.0500

Critical value = 1.974


Intervals for (column level mean) - (row level mean)

              1         2         3         4         5

     2   -6.000
          0.674

     3   -3.503    -0.885
          4.379     7.088

     4   -5.498    -2.887    -6.576
          1.298     4.014     1.499

     5  -11.152    -8.537   -12.187    -9.155
         -3.777    -1.065    -3.618    -1.574

     6   -8.611    -6.006    -9.751    -6.635    -1.580
         -2.538     0.184    -2.274    -0.314     5.360
```

MEN

Figure 56: ANOVA CALCULATED FREE TESTOSTEONE (ρMol/L)



Analysis of Variance on Calculated Free Testosterone

Source	DF	SS	MS	F	p
GRP	5	176169	35234	1.92	0.093
Error	167	3059126	18318		
Total	172	3235295			

Level	N	Mean	StDev
1	33	467.7	150.6
2	32	488.6	111.7
3	18	395.9	140.0
4	31	408.2	130.1
5	21	471.4	130.6
6	48	430.9	141.6

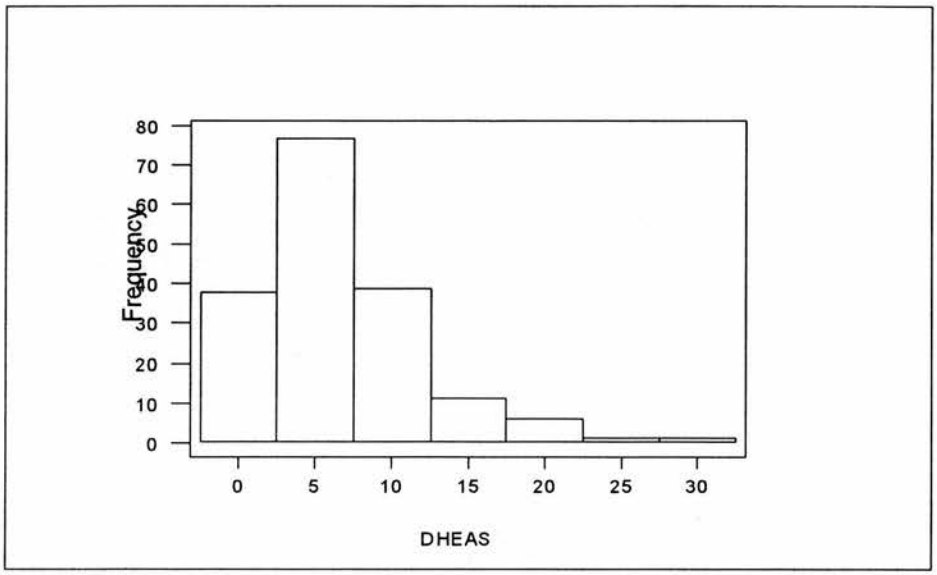
Pooled StDev = 135.3

Individual 95% CIs For Mean
Based on Pooled StDev

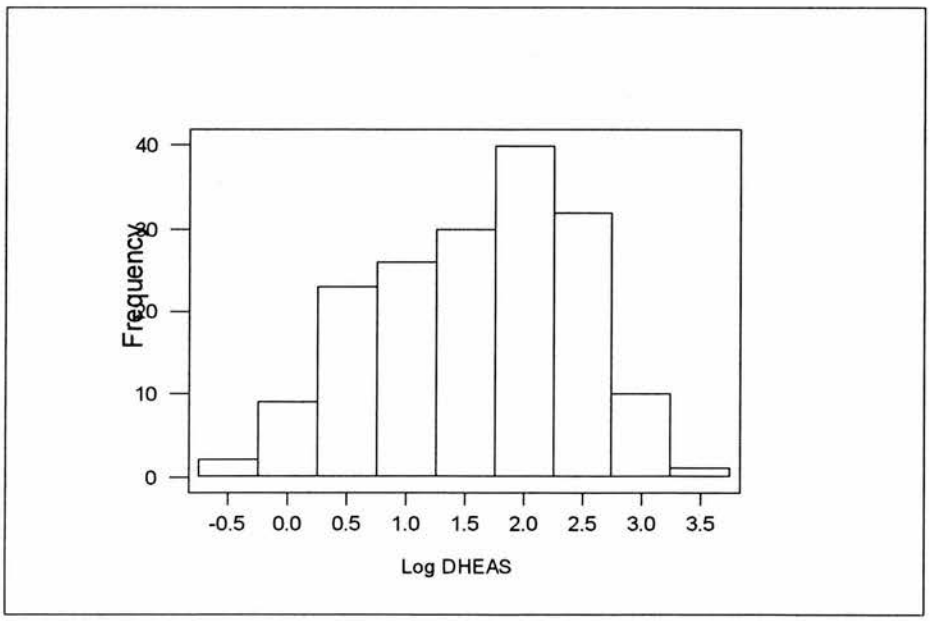
```
-----+---------+---------+---------+-
                  (-------*-------)
                     (-------*--------)
(----------*----------)
     (-------*-------)
                (---------*--------)
           (------*-----)
-----+---------+---------+---------+-
    360       420       480       540
```

MEN

Figure 57: ANOVA DEHYDROEPIANDOSTERONE SULPHATE (μMOL/L)



Because values not normally distributed logarithmic transformation performed

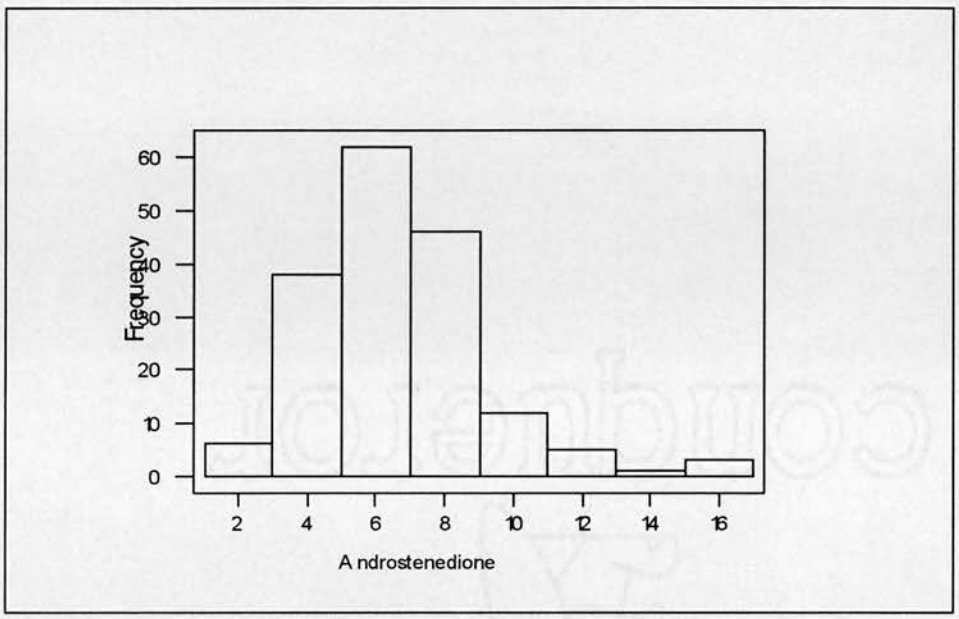


MEN

```
Analysis of Variance on log DHEAS
Source     DF        SS        MS        F        p
GRP         5    35.129     7.026    14.88    0.000
Error     181    78.844     0.472
Total     182   113.973

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level      N      Mean     StDev  ----+---------+---------+---------+--
     1     33    1.9406    0.8396                    (----*----)
     2     32    2.0742    0.6447                       (---*----)
     3     18    2.2000    0.5417                       (------*------)
     4     31    1.5162    0.7038            (----*----)
     5     21    1.2535    0.6873      (-----*-----)
     6     48    1.0067    0.6293  (---*---)
                                        ----+---------+---------+---------+--
Pooled StDev =    0.6871               1.00      1.50      2.00      2.50

Fisher's pairwise comparisons

    Family error rate = 0.362
Individual error rate = 0.0500

Critical value = 1.974

Intervals for (column level mean) - (row level mean)

               1         2         3         4         5

     2   -0.4783
          0.2111

     3   -0.6665   -0.5376
          0.1476    0.2859

     4    0.0734    0.2016    0.2668
          0.7753    0.9144    1.1008

     5    0.3061    0.4347    0.5040   -0.1289
          1.0680    1.2066    1.3890    0.6542

     6    0.6202    0.7478    0.8072    0.1830   -0.1116
          1.2475    1.3872    1.5794    0.8360    0.6053
```

MEN

Figure 58: ANOVA OF ANDROSTENEDIONE (ηMOL/L)



Analysis of Variance on Androstenedione

Source	DF	SS	MS	F	p
GRP	5	81.61	16.32	2.84	0.017
Error	180	959.14	5.74		
Total	181	1040.75			

Level	N	Mean	StDev	Individual 95% CIs For Mean Based on Pooled StDev
1	33	6.203	2.332	(------*------)
2	32	6.057	1.517	(------*-------)
3	18	5.906	2.030	(--------*---------)
4	31	6.107	2.140	(-------*------)
5	21	8.129	3.060	(--------*-------)
6	48	6.902	2.808	(-----*----)

Pooled StDev = 2.397

4.8 6.0 7.2 8.4

MEN

Fisher's pairwise comparisons

Family error rate = 0.362
Individual error rate = 0.0500

Critical value = 1.974

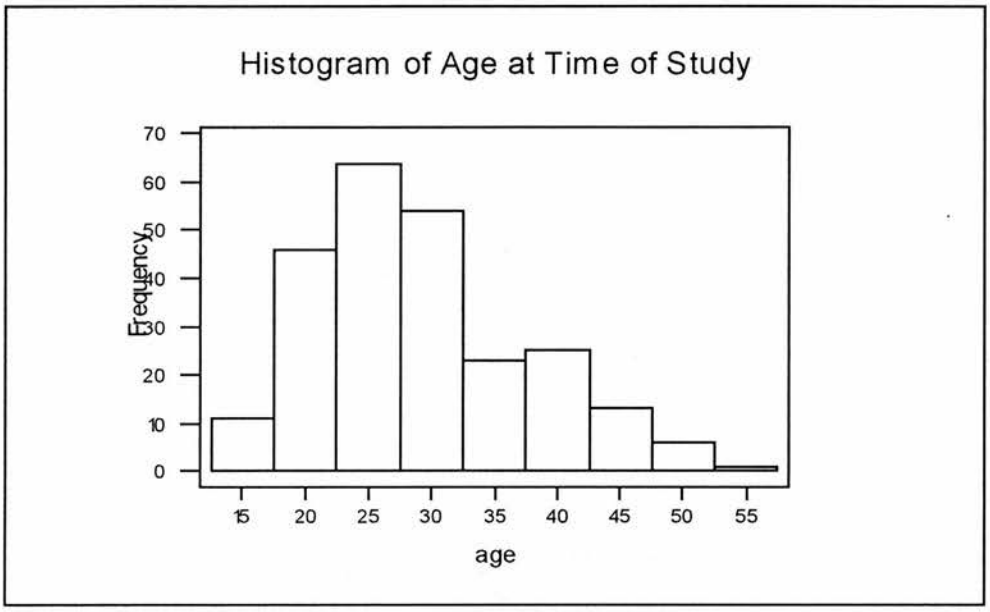
Intervals for (column level mean) - (row level mean)

	1	2	3	4	5
2	-1.056 1.349				
3	-1.123 1.717	-1.285 1.587			
4	-1.128 1.320	-1.294 1.193	-1.656 1.253		
5	-3.254 -0.597	-3.418 -0.726	-3.766 -0.679	-3.387 -0.656	
6	-1.793 0.395	-1.961 0.269	-2.343 0.350	-1.934 0.344	-0.024 2.477

APPENDIX FIVE : FEMALE STATISTICS

WOMEN

Figure 59: ANOVA OF AGE AT TIME OF STUDY




```
Analysis of Variance on age
Source     DF        SS        MS        F        p
group       2     566.5     283.2     4.06    0.018
Error     240   16753.5      69.8
Total     242   17320.0
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  -----+---------+---------+---------+-
     1     48    30.208     9.397                    (-------*-------)
     2     36    25.306     6.246  (--------*--------)
     3    159    29.252     8.434                    (----*---)
                                   -----+---------+---------+---------+-
Pooled StDev =    8.355              24.0      27.0      30.0      33.0
```

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

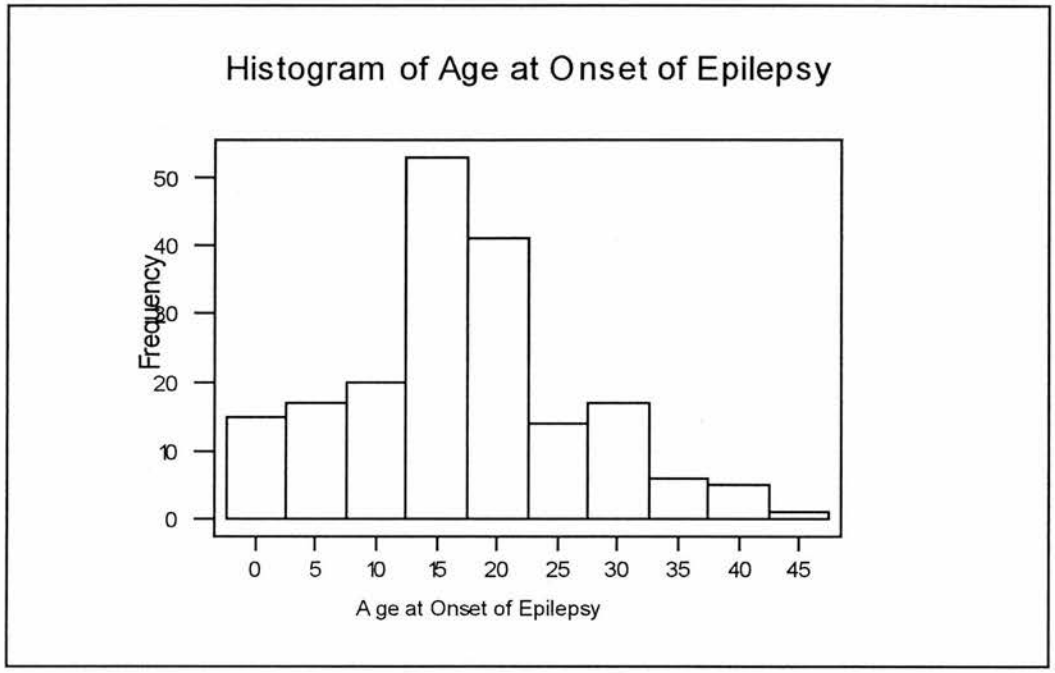
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

```
        1              2
  2  1.274
     8.532

  3 -1.754        -6.984
     3.667        -0.908
```

WOMEN

Figure 60:ANOVA OF AGE AT ONSET OF EPILEPSY




```
Analysis of Variance on age at onset of epilepsy
Source      DF        SS        MS         F         p
C91          1      77.9      77.9      0.92     0.339
Error      193   15865.1      84.8
Total      194   15943.0
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level       N      Mean     StDev  ----+---------+---------+---------+--
     2      36    18.437     7.427  (---------------*---------------)
     3     159    16.726     9.526  (-------*------)
                                    ----+---------+---------+---------+--
Pooled StDev =    9.211              16.0      18.0      20.0      22.0
```

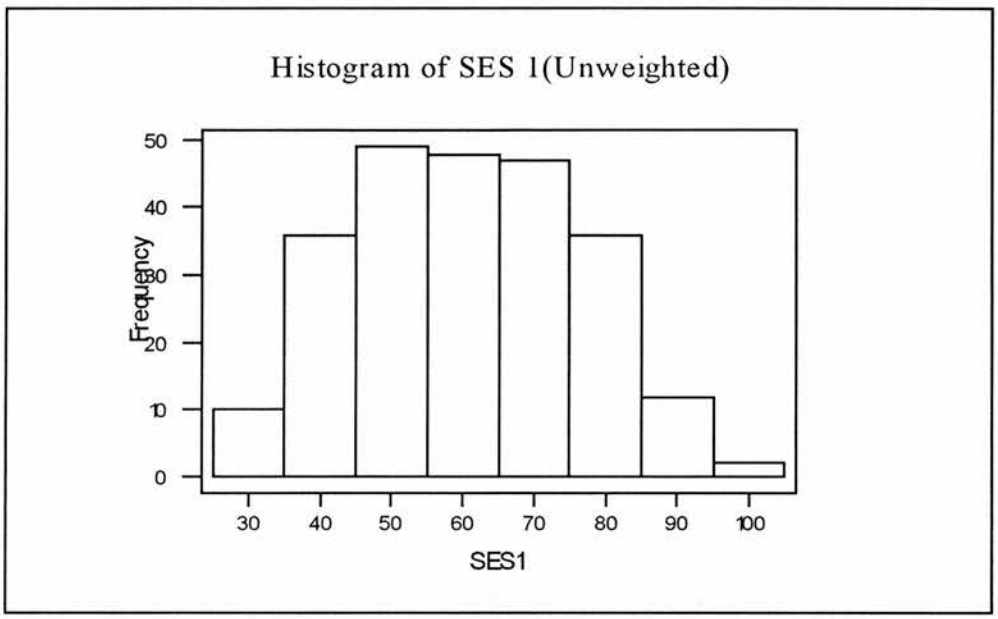
WOMEN

Figure 61: ANOVA OF AGE OF SEIZURE TYPE GROUPS

```
Analysis of Variance on age
Source     DF        SS        MS        F        p
cofit       3     149.0      49.7     0.70    0.555
Error     241   16684.2      71.3
Total     242   16833.1

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level      N      Mean     StDev  ----+---------+---------+---------+--
     1    110    29.340     8.389              (-------*-------)
     2     56    27.537     7.765  (-----------*----------)
     3     28    28.455     8.478    (--------------*--------------)
     4     48    29.600     9.292             (-----------*-----------)
                                    ----+---------+---------+---------+--
Pooled StDev =    8.444              26.0      28.0      30.0      32.0

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500
```

WOMEN

Figure 62:ANOVA OF SES 1(UNWEIGHTED)




```
Analysis of Variance on SES1
Source     DF        SS        MS        F        p
group       2      4307      2153     9.45    0.000
Error     237     53984       228
Total     239     58291
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  --+---------+---------+---------+----
     1     48     54.65     14.47     (-------*--------)
     2     36     54.33     16.33   (---------*---------)
     3    159     63.39     14.99                          (----*----)
                                   --+---------+---------+---------+----
Pooled StDev =    15.09           50.0      55.0      60.0      65.0
```

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

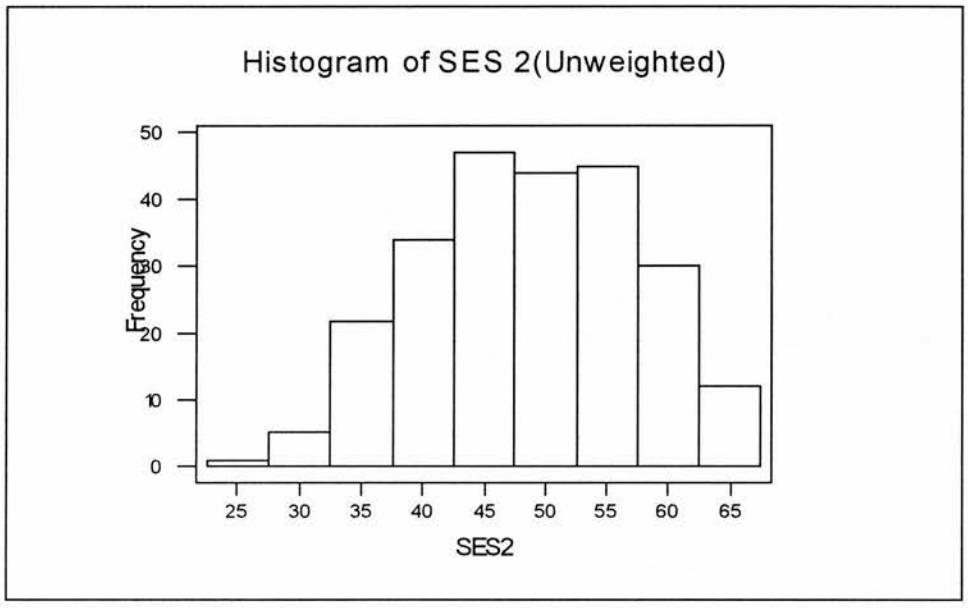
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-6.24 6.87	
3	-13.65 -3.84	-14.56 -3.56

WOMEN

Figure 63:ANOVA OF SES 2 (UNWEIGHTED)




```
Analysis of Variance on SES2
Source     DF        SS         MS         F         p
group       2    1014.0      507.0      6.74     0.001
Error     224   17819.8       75.2
Total     242   18833.7
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  --+---------+---------+---------+----
     1     48    44.687     7.360  (---------*---------)
     2     36    48.278     8.949               (----------*-----------)
     3    159    49.929     8.970                          (-----*----)
                                   --+---------+---------+---------+----
Pooled StDev =    8.671           42.5      45.0      47.5      50.0
```

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

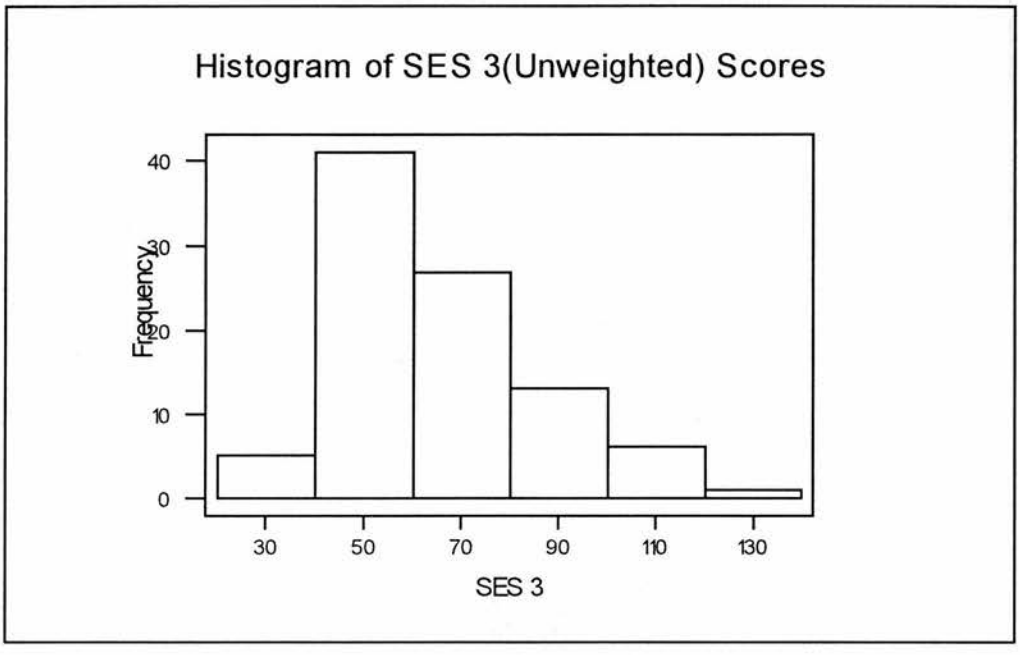
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-7.357 0.176	
3	-8.062 2.422	-4.810 1.507

WOMEN

Figure 64:ANOVA OF SES 3 (UNWEIGHTED)




```
Analysis of Variance on SES 3
Source      DF        SS         MS         F        p
MARRIED      2      3784       1892      5.04    0.008
Error       90     33770        375
Total       94     37554
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level       N      Mean     StDev  ---------+---------+---------+-------
     1      20     56.55     13.61          (--------*-------)
     2      10     53.30     19.21  (-----------*-----------)
     3      65     68.98     20.84                        (----*----)
                                    ---------+---------+---------+-------
Pooled StDev =     19.37                    50        60        70
```

Fisher's pairwise comparisons

Family error rate = 0.121

Individual error rate = 0.0500

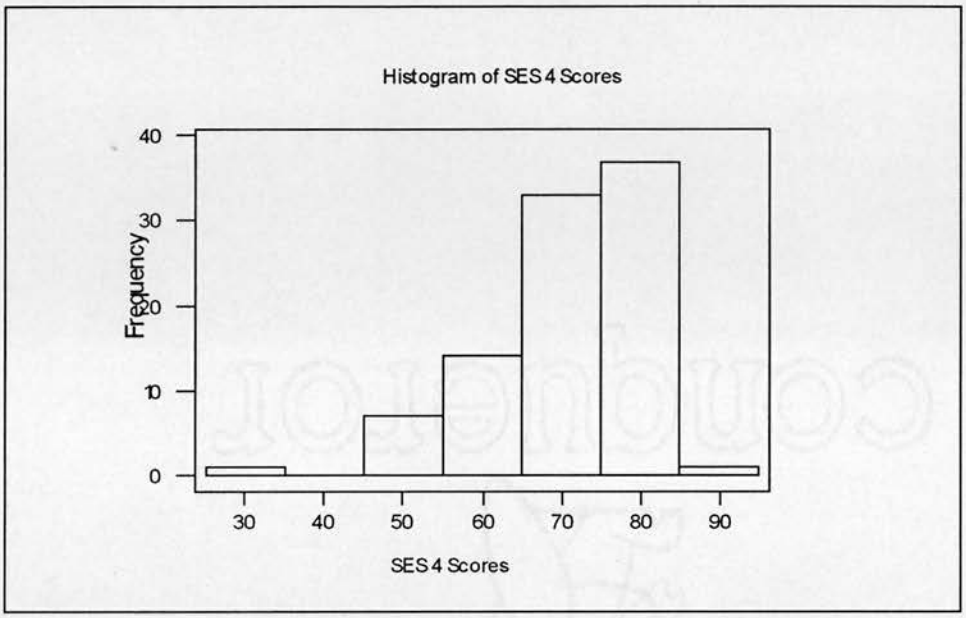
Critical value = 1.987

Intervals for (column level mean) - (row level mean)

```
            1         2

2     -11.66
       18.16

3     -22.31    -28.79
        2.56     -2.58
```

WOMEN

Figure 65:ANOVA OF SES 4 (UNWEIGHTED SCORES)



Analysis of Variance on SES4

Source	DF	SS	MS	F	p
group	2	458.8	229.4	2.46	0.091
Error	90	8391.0	93.2		
Total	94	8849.8			

Level	N	Mean	StDev
1	20	73.200	5.238
2	10	74.300	5.376
3	65	68.857	11.079

Pooled StDev = 9.656

Individual 95% CIs For Mean Based on Pooled StDev

```
----+---------+---------+---------+--
      (----------*----------)
     (--------------*--------------)
(-----*-----)
----+---------+---------+---------+--
 68.0      72.0      76.0      80.0
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

Critical value = 1.987

Intervals for (column level mean) - (row level mean)

	1	2
2	-8.531 6.331	
3	-0.581 9.267	-1.088 11.974

Because distribution not Gaussian analyisis repeated using a non parametric test

WOMEN

Kruskal-Wallis Test

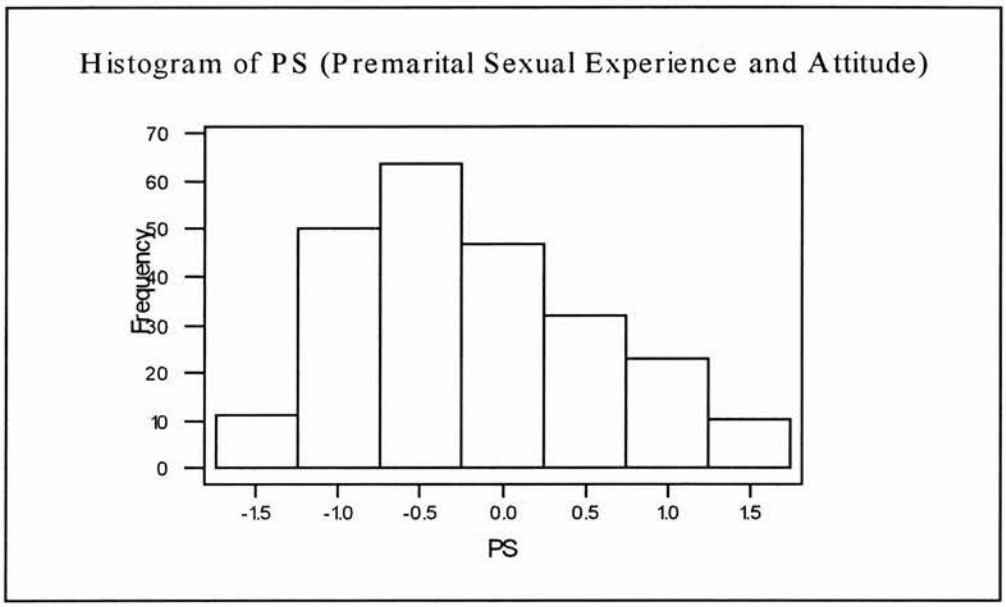
95 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	20	74.00	51.7	0.87
2	10	74.50	57.7	1.33
3	65	72.00	43.8	-1.64
OVERALL	95		47.0	

H = 3.04 d.f. = 2 p = 0.220
H = 3.04 d.f. = 2 p = 0.219 (adjusted for ties)

WOMEN

Figure 66:ANOVA OF PS (PREMARITAL SEXUAL EXPERIENCE AND ATTITUDE) SUBSCALE



Variable	N	Mean	Median	TrMean	StDev	SEMean
PS	243	-0.1854	-0.2900	-0.2097	0.7442	0.0483

Variable	Min	Max	Q1	Q3
PS	-1.5200	1.6300	-0.7800	0.3300

Analysis of Variance on PS

Source	DF	SS	MS	F	p
group	2	3.963	1.982	3.66	0.027
Error	240	126.758	0.542		
Total	242	130.721			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev
1	48	-0.3240	0.7227
2	36	-0.4039	0.7754
3	159	-0.0921	0.7307

Pooled StDev = 0.7360

WOMEN

Fisher's pairwise comparisons

Family error rate = 0.122

individual error rate = 0.0500

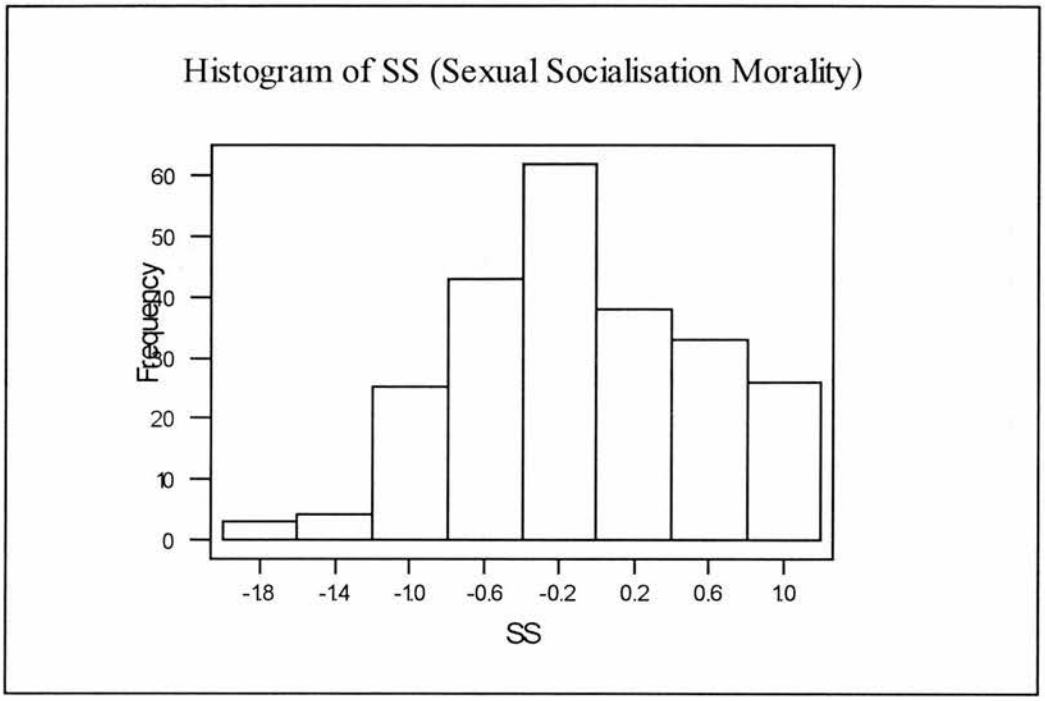
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.2413 0.4010	
3	-0.4736 0.0097	-0.5802 -0.0434

WOMEN

Figure 67:ANOVA OF SS (SEXUAL SOCIALISATION MORALITY) SUBSCALE



Analysis of Variance on SS

Source	DF	SS	MS	F	p
group	2	6.550	3.275	8.77	0.000
Error	240	86.264	0.373		
Total	242	92.814			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-------+---------+---------+---------
1	48	-0.3104	0.5258	(-------*--------)
2	36	-0.3211	0.5894	(---------*---------)
3	159	0.0346	0.6397	(----*----)
				-------+---------+---------+---------
Pooled StDev =		0.6111		-0.40 -0.20 -0.00

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

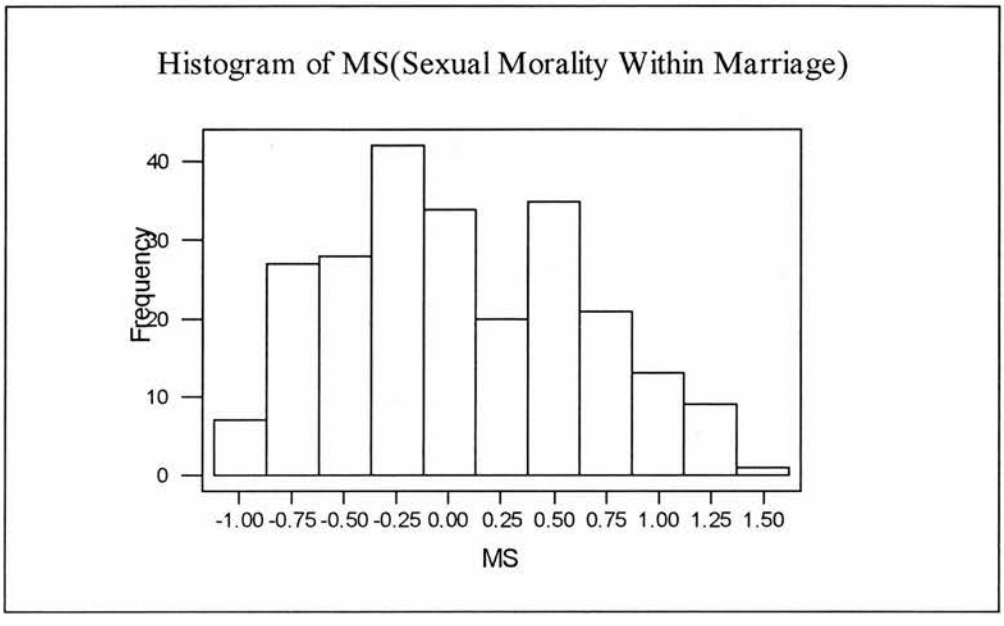
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.2559 0.2773	
3	-0.5461 -0.1439	-0.5790 -0.1324

WOMEN

Figure 68:ANOVA OF MS (SEXUAL MORALITY WITHIN MARRIAGE) SUBSCALE



Analysis of Variance on MS

Source	DF	SS	MS	F	p
group	2	4.941	2.471	7.63	0.001
Error	240	75.761	0.324		
Total	242	80.702			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-+---------+---------+---------+-----
1	48	-0.1609	0.5537	(---------*---------)
2	36	-0.1133	0.6505	(-----------*-----------)
3	159	0.1610	0.5534	(-----*-----)
				-+---------+---------+---------+-----
Pooled StDev =		0.5690		-0.32 -0.16 0.00 0.16

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

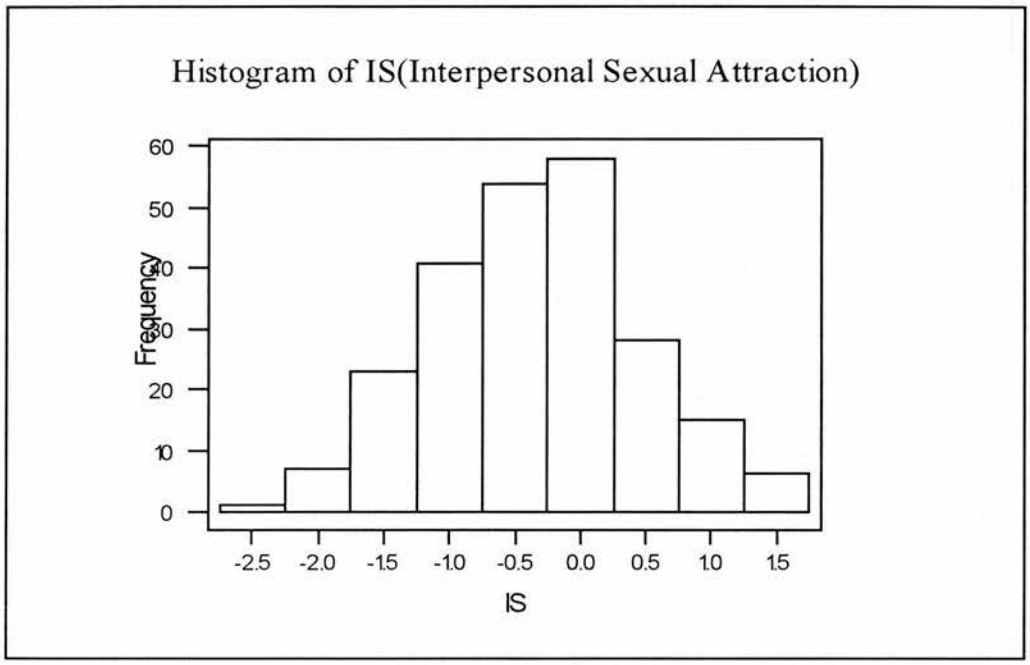
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.2958 0.2007	
3	-0.5087 0.1351	-0.4819 -0.0669

WOMEN

Figure 69:ANOVA OF IS (INTERPERSONAL SEXUAL ATTRACTION) SUBSCALE



Analysis of Variance on IS

Source	DF	SS	MS	F	p
group	2	6.682	3.341	5.78	0.004
Error	240	132.939	0.578		
Total	242	139.621			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-----+---------+---------+---------+-
1	48	-0.6459	0.5853	(--------*--------)
2	36	-0.4917	0.8122	(---------*---------)
3	159	-0.2364	0.7936	(----*---)
				-----+---------+---------+---------+-
Pooled StDev =		0.7603		-0.75 -0.50 -0.25 0.00

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

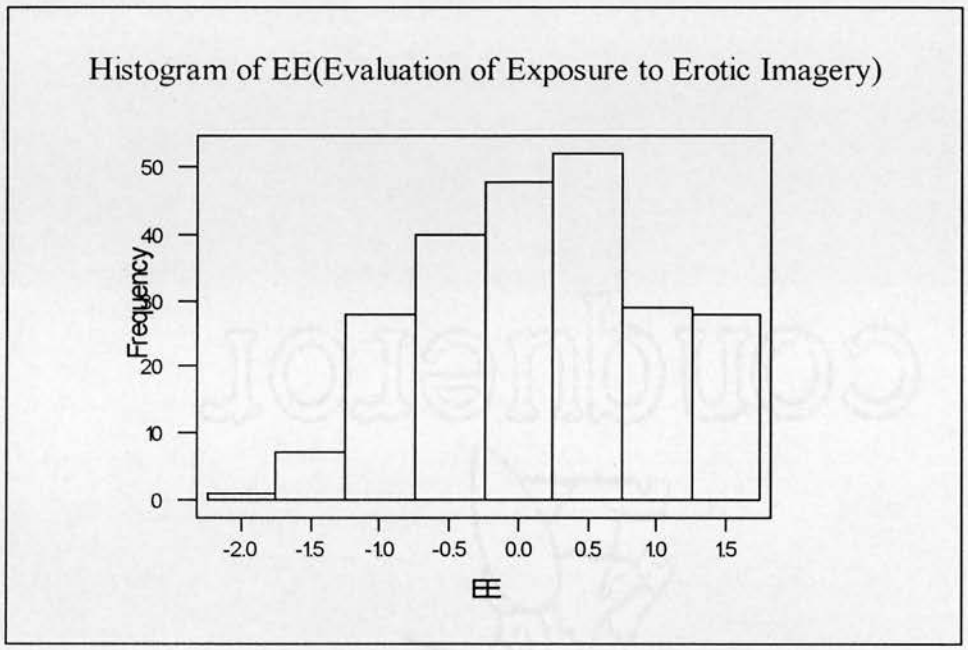
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.4901 0.1818	
3	-0.6615 0.1574	-0.5361 0.0255

WOMEN

Figure 70:ANOVA OF EE (EVALUATION OF EXPOSURE TO EROTIC IMAGERY) SUBSCALE



Analysis of Variance on EE

Source	DF	SS	MS	F	p
group	2	6.024	3.012	4.79	0.009
Error	240	144.534	0.628		
Total	242	150.558			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	----------+---------+---------+---------
1	48	-0.1822	0.7696	(---------*--------)
2	36	0.1025	0.8181	(---------*----------)
3	159	0.2301	0.7936	(----*----)

Pooled StDev = 0.7927 ----------+---------+---------+---------
-0.25 0.00 0.25

Fisher's pairwise comparisons

Family error rate = 0.122

Individual error rate = 0.0500

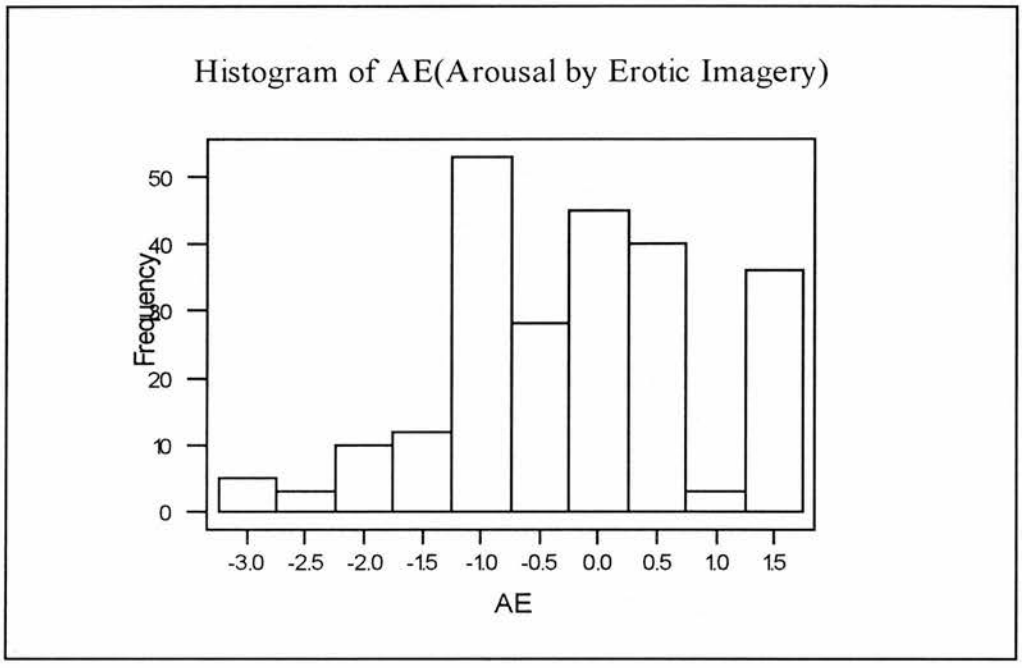
Critical value = 1.970

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.6322 0.0628	
3	-0.6753 -0.1493	-0.4173 0.1620

WOMEN

Figure 71:ANOVA OF AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE



Analysis of Variance on AE

Source	DF	SS	MS	F	p
group	2	3.83	1.91	1.70	0.185
Error	240	261.02	1.13		
Total	242	264.85			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-------+---------+---------+---------
1	48	-0.360	0.965	(----------*----------)
2	36	-0.322	1.206	(----------*----------)
3	159	-0.078	1.053	(------*------)
				-------+---------+---------+---------
Pooled StDev =		1.061		-0.50 -0.25 0.00

Because the distribution of AE scores is skewed and it was not possible to correct this by logarithmic transformation the analysis was checked using Kruskal-Wallis

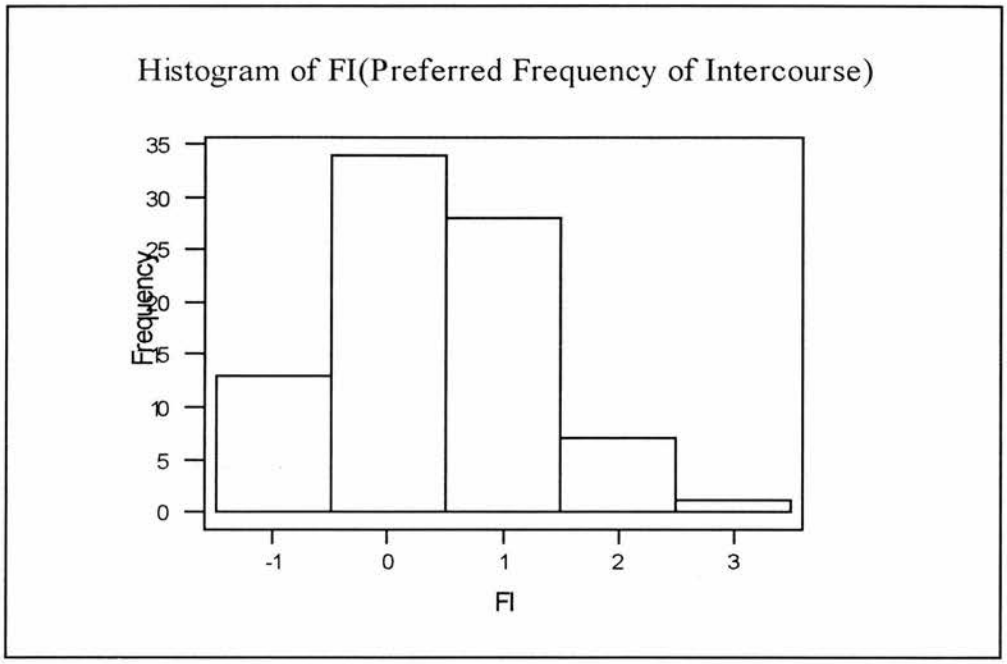
243 ces were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	48	-0.3800	104.0	-1.58
2	36	-0.3800	112.3	-0.55
3	159	0.1600	123.7	1.73
OVERALL	243		118.0	

H = 3.31 d.f. = 2 p = 0.191
H = 3.40 d.f. = 2 p = 0.184 (adjusted for ties)

WOMEN

Figure 72:ANOVA OF FI (PREFERRED FREQUENCY OF INTERCOURSE) SUBSCALE



Analysis of Variance on FI

Source	DF	SS	MS	F	p
MARRIED	2	3.777	1.888	2.71	0.073
Error	93	55.723	0.697		
Total	94	59.500			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-+---------+---------+---------+-----
1	20	0.5832	0.6133	(----------*----------)
2	10	0.7360	0.8488	(--------------*--------------)
3	65	0.1976	0.8952	(------*-----)
				-+---------+---------+---------+-----
Pooled StDev =		0.8346		0.00 0.35 0.70 1.05

Fisher's pairwise comparisons

Family error rate = 0.121

Individual error rate = 0.0500

Critical value = 1.990

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.8017 0.4960	
3	-0.0575 0.8286	-0.0334 1.1102

WOMEN

Because the distribution of FI scores was skewed it was checked using the Kruskall-Wallis

Kruskal-Wallis Test

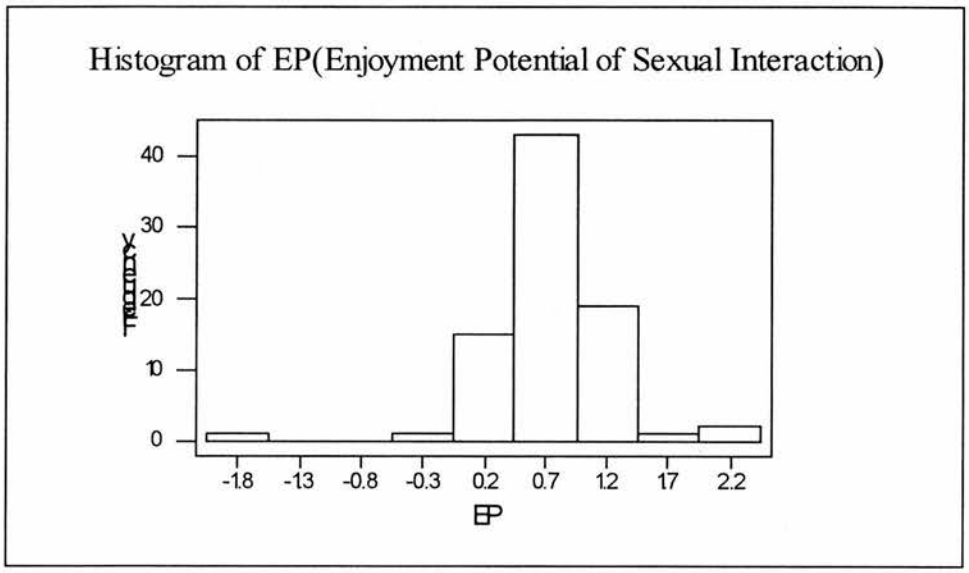
95 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	20	0.6300	50.1	1.67
2	10	0.5850	52.6	1.48
3	65	0.1015	37.2	-2.48
OVERALL	95		42.0	

H = 6.24 d.f. = 2 p = 0.045
H = 6.24 d.f. = 2 p = 0.045 (adjusted for ties)

WOMEN

Figure 73:ANOVA OF EP (ENJOYMENT POTENTIAL OF SEXUAL INTERACTION) SUBSCALE



Analysis of Variance on EP

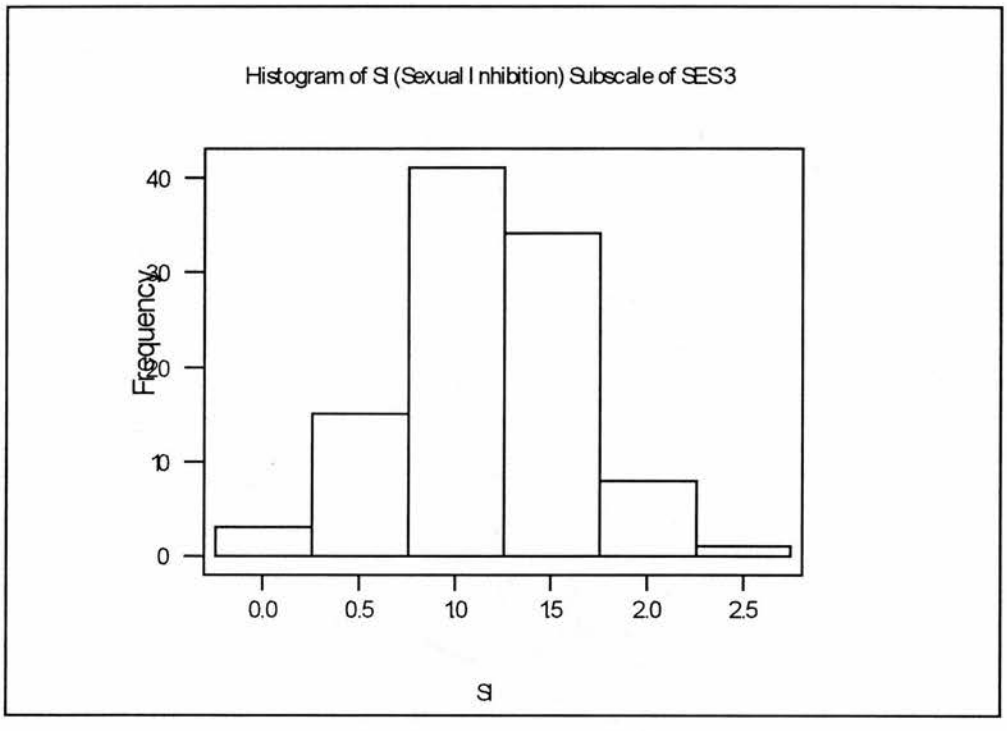
Source	DF	SS	MS	F	p
MARRIED	2	0.779	0.389	1.60	0.209
Error	93	19.258	0.244		
Total	94	20.037			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	---+---------+---------+---------+---
1	20	0.8516	0.3971	(--------*--------)
2	10	0.5070	0.8238	(-----------*-----------)
3	65	0.7392	0.4453	(-----*----)
				---+---------+---------+---------+---
Pooled StDev =		0.4937		0.25 0.50 0.75 1.00

WOMEN

Figure 74:ANOVA OF SI (SEXUAL INHIBITION) SUBSCALE OF SES 3



Analysis of Variance on SI

Source	DF	SS	MS	F	p
GRPS	2	0.926	0.463	2.26	0.110
Error	92	20.315	0.205		
Total	93	21.242			

Level	N	Mean	StDev
1	20	0.9954	0.3926
2	10	1.3050	0.3601
3	65	1.1670	0.4893

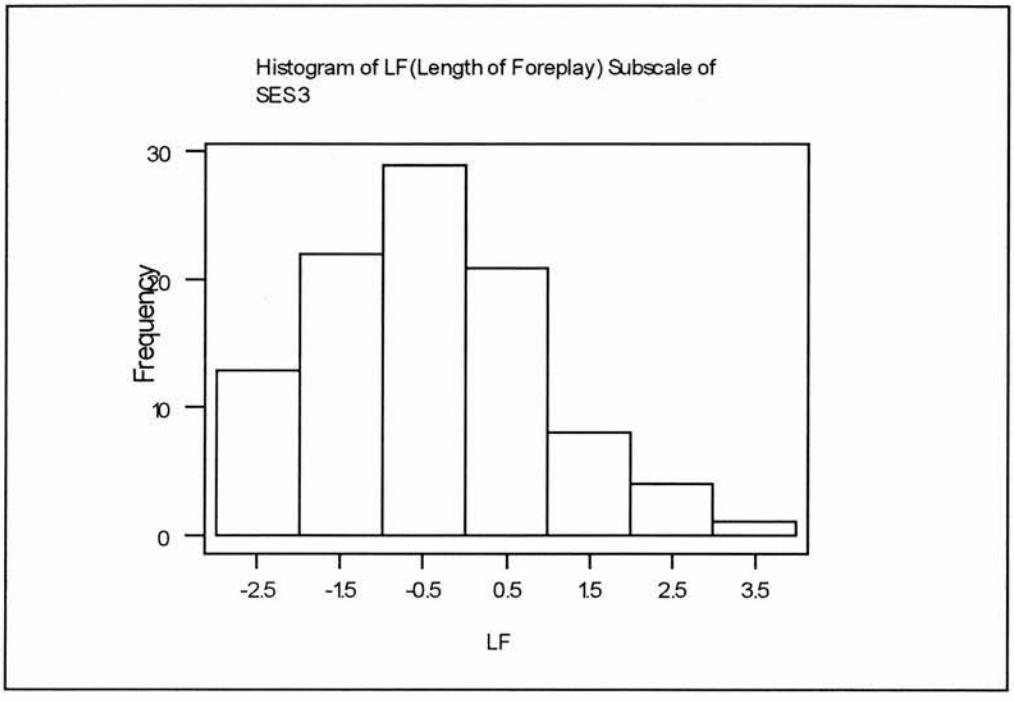
Pooled StDev = 0.4530

Individual 95% CIs For Mean
Based on Pooled StDev

```
---------+---------+---------+------
 (--------*--------)
              (-----------*-----------)
              (----*-----)
---------+---------+---------+------
       1.00      1.20      1.40
```

WOMEN

Figure 75:ANOVA OF LF (LENGTH OF FOREPLAY) SUBSCALE OF SES 3



Analysis of Variance on LF

Source	DF	SS	MS	F	p
GRPS	2	4.32	2.16	1.18	0.313
Error	92	174.33	1.84		
Total	93	178.65			

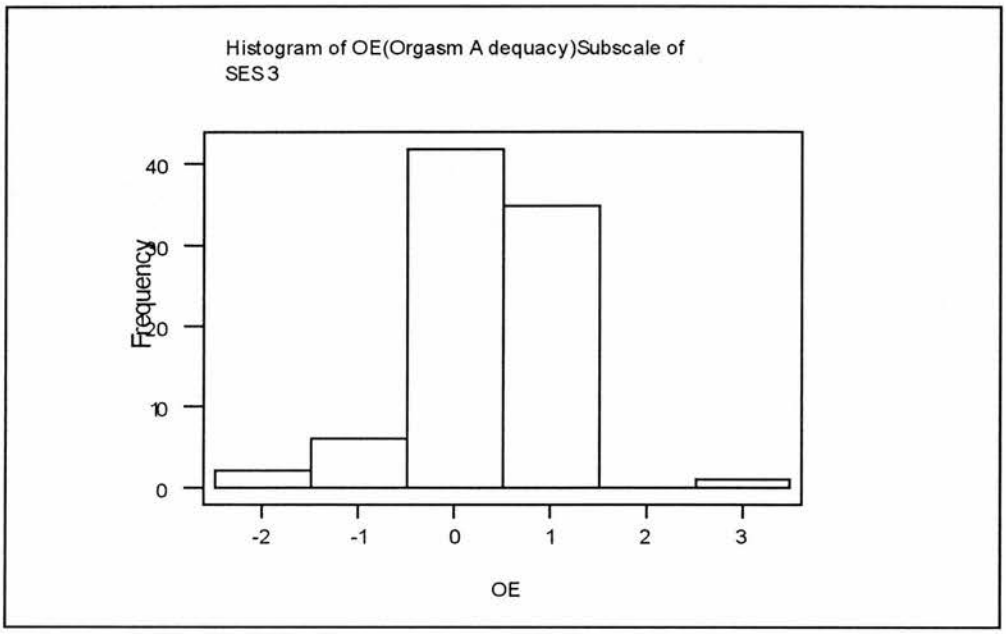
Level	N	Mean	StDev
1	20	-0.465	1.251
2	10	0.004	1.599
3	65	-0.627	1.338
Pooled StDev =		1.355	

Individual 95% CIs For Mean Based on Pooled StDev

```
-+---------+---------+---------+-----
 (-----------*----------)
          (--------------*--------------)
    (-----*------)
-+---------+---------+---------+-----
-1.00     -0.50      0.00      0.50
```

WOMEN

Figure 76:ANOVA OF OE (ORGASM ADEQUACY DURING INTERCOURSE AND ITS EVALUATION) SUBSCALE OF SES 3



Analysis of Variance on OE

Source	DF	SS	MS	F	p
GRPS	2	4.304	2.152	4.58	0.013
Error	92	38.995	0.470		
Total	93	43.299			

Level	N	Mean	StDev
1	20	-0.0805	0.4639
2	10	-0.0323	0.8321
3	65	0.4057	0.7160

Pooled StDev = 0.6854

Individual 95% CIs For Mean Based on Pooled StDev

```
-----+---------+---------+---------+-
  (---------*----------)
(-------------*-------------)
                        (------*-----)
-----+---------+---------+---------+-
  -0.30      0.00      0.30      0.60
```

Fisher's pairwise comparisons

Family error rate = 0.121
Individual error rate = 0.0500

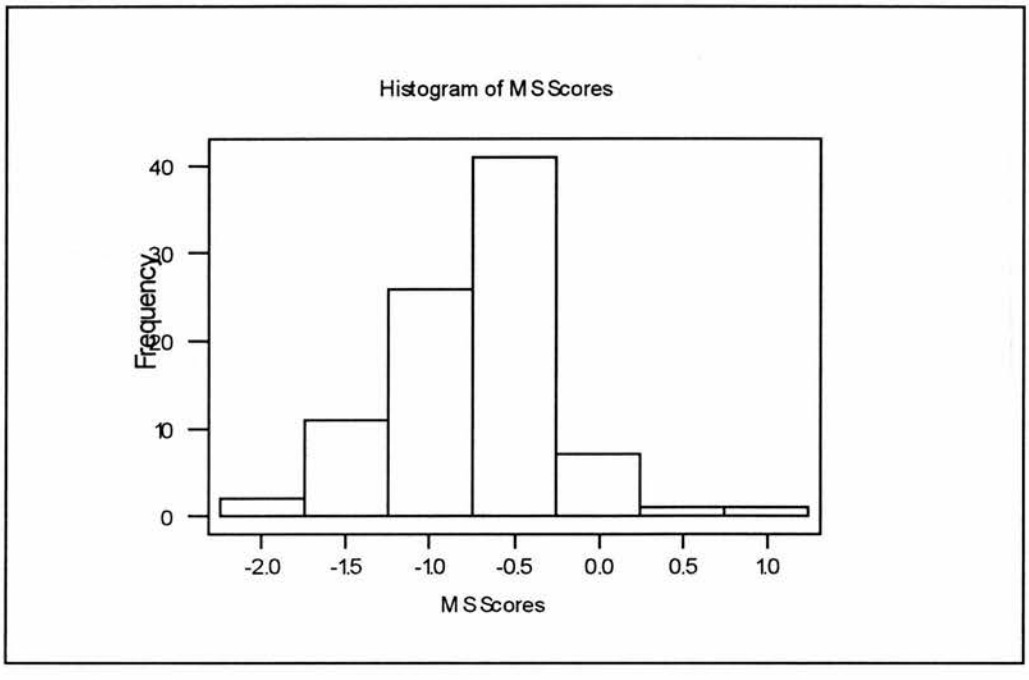
Critical value = 1.989

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.5648 0.4683	
3	-0.8481 -0.1242	-0.8876 0.0117

WOMEN

Figure 77:ANOVA OF MS(MARITAL SATISFACTION) SUBSCALE OF SES 4



Analysis of Variance on MS

Source	DF	SS	MS	F	p
group	2	0.411	0.206	0.94	0.394
Error	93	18.797	0.219		
Total	94	19.209			

Level	N	Mean	StDev
1	20	-0.6525	0.3131
2	10	-0.6250	0.4879
3	65	-0.7863	0.5050

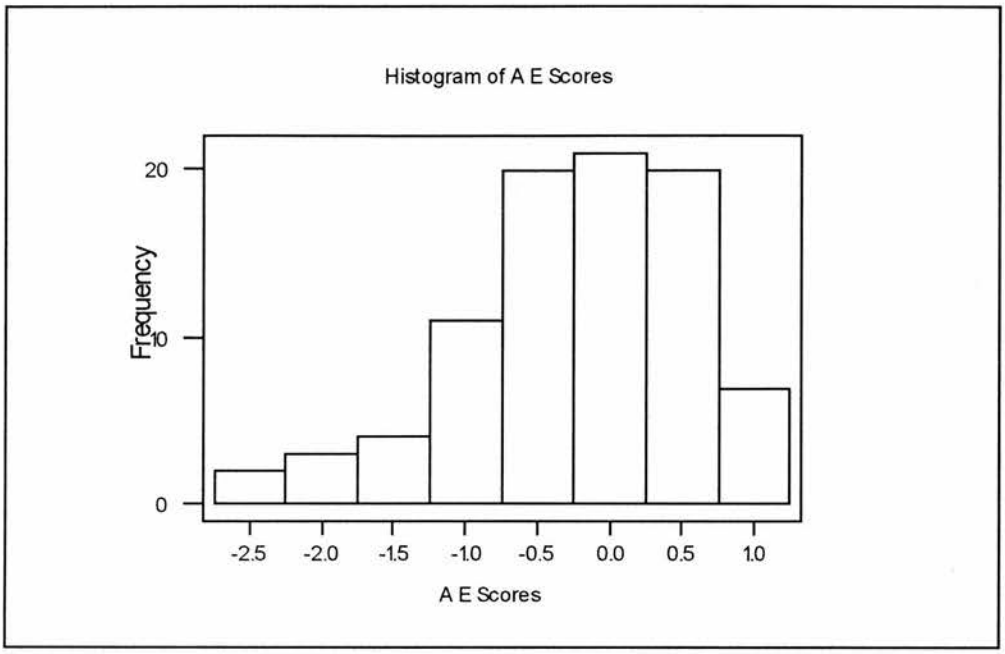
Pooled StDev = 0.4675

Individual 95% CIs For Mean
Based on Pooled StDev

```
--------+---------+---------+--------
     (------------*------------)
   (------------------*-----------------)
   (-------*------)
--------+---------+---------+--------
      -0.80     -0.64     -0.48
```

WOMEN

Figure 78:ANOVA OF AE(ATTITUDE TOWARDS EXTRAMARITAL INVOLVEMENT)SUBSCALE OF SES 4




```
Analysis of Variance on AE
Source     DF        SS         MS         F         p
group       2     4.903      2.451      4.17     0.019
Error      93    49.985      0.588
Total      94    54.888

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level      N      Mean     StDev  ---------+---------+---------+-------
     1     20    0.0740    0.5010            (---------*---------)
     2     10    0.0290    0.7769       (-------------*-------------)
     3     65   -0.4383    0.8354  (----*-----)
                                    ---------+---------+---------+-------
Pooled StDev =    0.7668               -0.35     -0.00      0.35

Fisher's pairwise comparisons

    Family error rate = 0.121
Individual error rate = 0.0500

Critical value = 1.988

Intervals for (column level mean) - (row level mean)

               1         2

     2   -0.5454
          0.6354

     3    0.1170   -0.0547
          0.9076    0.9893
```

WOMEN

Because the distribution of values is not Gaussian the analysis was used using a non-parametric test.

Kruskal-Wallis Test

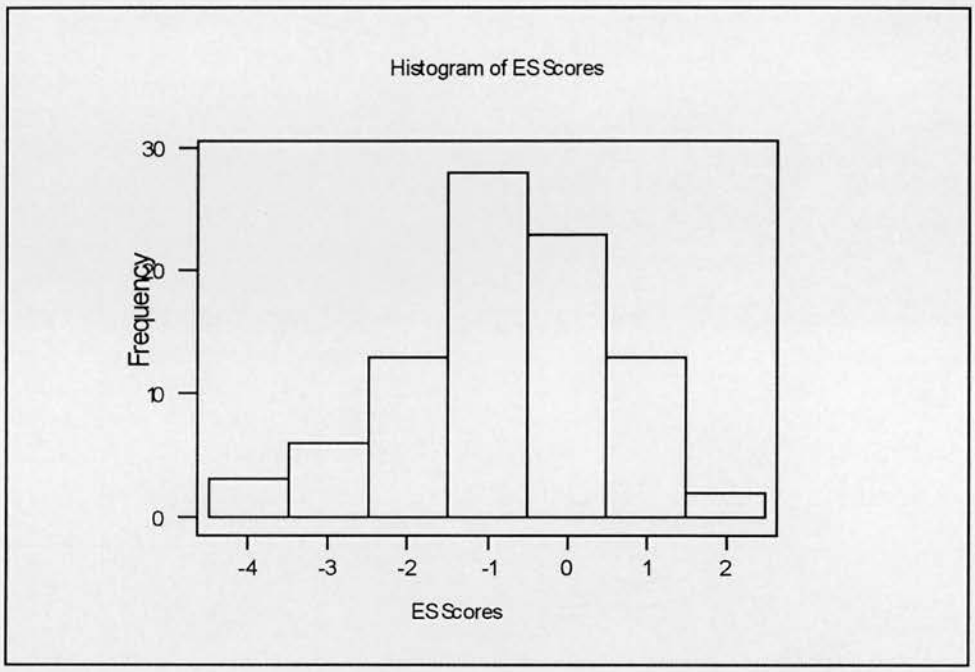
95 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	20	0.1850	55.2	2.14
2	10	0.4300	54.3	1.29
3	65	-0.3300	39.1	-2.75
OVERALL	95		44.5	

H = 7.58 d.f. = 2 p = 0.023
H = 7.58 d.f. = 2 p = 0.023 (adjusted for ties)

WOMEN

Figure 79:ANOVA OF ES(EVALUATION OF PARTNER AS SEXUAL PARTNER)




```
Analysis of Variance on ES
Source     DF        SS         MS         F         p
group       2     10.48       5.24      3.40     0.038
Error      93    130.86       1.54
Total      94    141.34

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level      N      Mean     StDev  ---+---------+---------+---------+---
     1     20    -0.474     0.996         (--------*--------)
     2     10    -0.102     1.336           (------------*------------)
     3     65    -1.045     1.297  (-----*----)
                                   ---+---------+---------+---------+---
Pooled StDev =     1.241           -1.20     -0.60      0.00      0.60

Fisher's pairwise comparisons

    Family error rate = 0.121
Individual error rate = 0.0500

Critical value = 1.988

Intervals for (column level mean) - (row level mean)

              1         2

     2    -1.327
           0.583

     3    -0.068     0.099
           1.211     1.788
```

WOMEN

Figure 80:ANOVA OF PS(PREMARITAL SEXUAL EXPERIENCE) SUBSCALE OF SES 1 FOR SEIZURE TYPE GROUPS

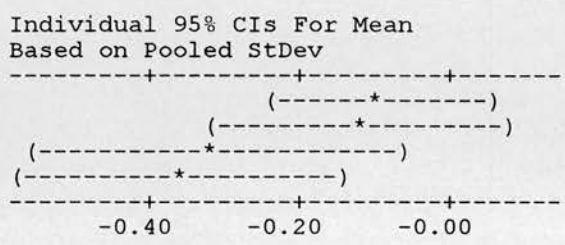
Analysis of Variance on PS

Source	DF	SS	MS	F	p
cofit	3	2.915	0.972	1.77	0.154
Error	241	125.432	0.550		
Total	242	128.346			

Level	N	Mean	StDev
CPS	110	-0.0936	0.8071
PGE	56	-0.1196	0.6910
UND	28	-0.3130	0.6636
CONT	48	-0.3566	0.6912

Pooled StDev = 0.7417

Individual 95% CIs For Mean Based on Pooled StDev



CPS Complex Partial Seizures
PGE Primary Generalised Seizures
Und Undefined Seizures
Cont Control

Figure 81:ANOVA OF SS(SEXUAL SOCIALISATION) SUBSCALE OF SES 1FOR SEIZURE TYPE GROUPS

```
One-Way Analysis of Variance

Analysis of Variance on SS
Source     DF        SS        MS        F        p
cofit       3     2.535     0.845     2.15    0.095
Error     241    88.539     0.394
Total     242    91.074

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level      N      Mean     StDev  -----+---------+---------+---------+-
 CPS      110   -0.0406    0.6532                    (-----*-----)
 PGE       56   -0.0450    0.6295                 (--------*-------)
 UND       28   -0.0787    0.6509              (----------*----------)
 CONT      48   -0.3134    0.5398  (---------*---------)
                                      -----+---------+---------+---------+-
Pooled StDev =    0.6273               -0.40     -0.20     -0.00      0.20

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.971

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.2063
          0.2151

     3   -0.2155   -0.2468
          0.2917    0.3143

     4    0.0498    0.0151   -0.0552
          0.4958    0.5217    0.5246


CPS Complex Partial Seizures
PGE Primary Generalised Seizures
Und Undefined Seizures
Cont Control
```

WOMEN

Figure 82:ANOVA OF MS (SEXUAL MORALITY WITHIN MARRIAGE) SUBSCALE OF SES 1FOR SEIZURE TYPE GROUPS

One-Way Analysis of Variance

```
Analysis of Variance on MS
Source     DF        SS        MS        F        p
cofit       3     3.861     1.287     3.96    0.009
Error     241    74.083     0.325
Total     242    77.944

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level      N      Mean     StDev  --------+---------+---------+--------
 CPS      110    0.1653    0.6220                      (----*-----)
 PGE       56    0.0463    0.5002               (------*-------)
 UND       28    0.0630    0.5361             (---------*---------)
 CONT      48   -0.1882    0.5428  (--------*-------)
                                   --------+---------+---------+--------
Pooled StDev =   0.5700                 -0.20      0.00      0.20

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.970

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.0720
          0.3100

     3   -0.1223   -0.2666
          0.3269    0.2332

     4    0.1513    0.0045   -0.0074
          0.5558    0.4645    0.5098


CPS Complex Partial Seizures
PGE Primary Generalised Seizures
Und Undefined Seizures
Cont Control
```

WOMEN

Figure 83:ANOVA OF EE (EVALUATION OF EXPOSURE TO EROTIC IMAGERY) SUBSCALE OF SES 2 FOR DIFFERENT SEIZURE TYPE GROUPS

Analysis of Variance on EE

```
Source      DF        SS        MS        F        p
cofit        3     8.667     2.889     4.68    0.003
Error      241   138.410     0.618
Total      242   147.077

                                    Individual 95% CIs For Mean
                                    Based on Pooled StDev
 Level       N      Mean     StDev  --------+---------+---------+--------
 CPS       110    0.3212    0.7359                            (-----*-----)
 PGE        56    0.0275    0.9102              (-------*--------)
 UND        28    0.1216    0.7270               (----------*----------)
 CONT       48   -0.1895    0.7809   (--------*---------)
                                    --------+---------+---------+--------
Pooled StDev =    0.7861                 -0.25      0.00      0.25

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.971

Intervals for (column level mean) - (row level mean)

               1         2         3

     2    0.0297
          0.5577

     3   -0.1182   -0.4457
          0.5173    0.2575

     4    0.2290   -0.1023   -0.0539
          0.7924    0.5364    0.6762


CPS Complex Partial Seizures
PGE Primary Generalised Seizures
Und Undefined Seizures
Cont Control
```

WOMEN

Figure 84:ANOVA OF IS (INTERPERSONAL SEXUAL ATTRACTION) SUBSCALE OF SES 2 FOR DIFFERENT SEIZURE TYPE GROUPS

```
One-Way Analysis of Variance

Analysis of Variance on IS
Source     DF        SS        MS        F        p
cofit       3     7.251     2.417     4.19    0.007
Error     241   129.132     0.576
Total     242   136.383

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level      N      Mean     StDev  ------+---------+---------+---------+
  CPS     110   -0.1999    0.7856                          (-----*-----)
  PGE      56   -0.4226    0.8413                (-------*--------)
  UND      28   -0.2379    0.7530                     (---------*----------)
  CONT     48   -0.6658    0.5768  (--------*--------)
                                      ------+---------+---------+---------+
Pooled StDev =   0.7593                -0.75     -0.50     -0.25      0.00

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.971

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.0357
          0.4811

     3   -0.2617   -0.5204
          0.3377    0.1509

     4    0.1938   -0.0680    0.0816
          0.7380    0.5545    0.7743

CPS Complex Partial Seizures
PGE Primary Generalised Seizures
Und Undefined Seizures
Cont Control
```

WOMEN

Figure 85:ANOVA OF AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE OF SES 2 FOR SEIZURE TYPE GROUPS

Analysis of Variance on AE

```
Source      DF        SS        MS        F        p
cofit        3      5.03      1.68     1.47    0.224
Error      240    259.07      1.14
Total      241    264.10

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  -------+---------+---------+---------
 CPS       110    -0.010     1.029                    (--------*-------)
 PGE        56    -0.292     1.186       (----------*----------)
 UND        28    -0.213     1.138       (--------------*--------------)
 CONT       48    -0.359     0.958  (------------*-----------)
                                      -------+---------+---------+---------
Pooled StDev =     1.068              -0.50     -0.25      0.00
```

CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control

WOMEN

Figure 86:ANOVA OF EE (EVALUATION OF EXPOSURE TO EROTIC IMAGERY) SUBSCALE OF SES 2 FOR SEIZURE TYPE GROUPS

Analysis of Variance on EE

```
Source      DF        SS        MS        F        p
cofit        3     8.667     2.889     4.68    0.003
Error      240   138.410     0.618
Total      241   147.077

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  --------+---------+---------+--------
  CPS      110    0.3212    0.7359                              (-----*-----)
  PGE       56    0.0275    0.9102                (-------*--------)
  UND       28    0.1216    0.7270                 (----------*----------)
  CONT      48   -0.1895    0.7809   (--------*---------)
                                      --------+---------+---------+--------
Pooled StDev =    0.7861                  -0.25      0.00      0.25

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.971

Intervals for (column level mean) - (row level mean)

               1         2         3

     2    0.0297
          0.5577

     3   -0.1182   -0.4457
          0.5173    0.2575

     4    0.2290   -0.1023   -0.0539
          0.7924    0.5364    0.6762


CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control
```

WOMEN

Figure 87:ANOVA OF IS (INTERPERSONAL SEXUAL ATTRACTION) SUBSCALE OF SES 2 FOR SEIZURE TYPE GROUPS

Analysis of Variance on IS

```
Source      DF         SS         MS         F         p
cofit        3      7.251      2.417      4.19     0.007
Error      240    129.132      0.576
Total      241    136.383

                                        Individual 95% CIs For Mean
                                        Based on Pooled StDev
 Level       N       Mean      StDev  ------+---------+---------+---------+
 CPS       110    -0.1999     0.7856                        (-----*-----)
 PGE        56    -0.4226     0.8413              (-------*--------)
 UND        28    -0.2379     0.7530                   (---------*----------)
 CONT       48    -0.6658     0.5768  (--------*--------)
                                       ------+---------+---------+---------+
Pooled StDev =    0.7593                 -0.75     -0.50     -0.25      0.00

Fisher's pairwise comparisons

    Family error rate = 0.202
Individual error rate = 0.0500

Critical value = 1.971

Intervals for (column level mean) - (row level mean)

               1         2         3

     2    -0.0357
           0.4811

     3    -0.2617   -0.5204
           0.3377    0.1509

     4     0.1938   -0.0680    0.0816
           0.7380    0.5545    0.7743


CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control
```

WOMEN

Figure 88:ANOVA OF FI (PREFERRED FREQUENCY OF INTERCOURSE) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on FI

Source	DF	SS	MS	F	p
cofit	3	1.838	0.613	0.84	0.474
Error	93	59.628	0.727		
Total	94	61.466			

Level	N	Mean	StDev
CPS	43	0.2366	1.0254
PGE	16	0.2205	0.6962
UND	16	0.3225	0.7239
CONT	20	0.5994	0.6268

Pooled StDev = 0.8527

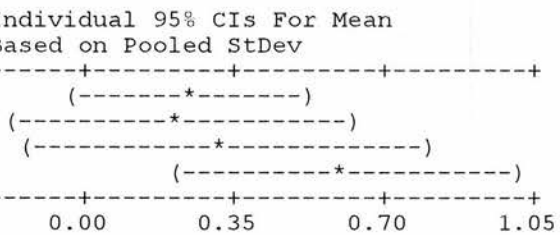


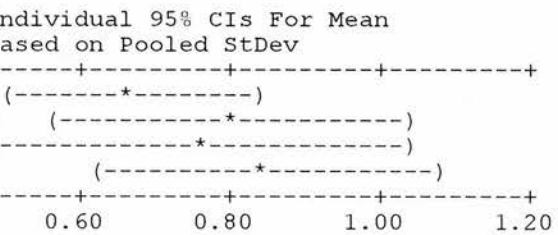
Figure 89:ANOVA OF EP (ENJOYMENT POTENTIAL OF INTERCOURSE) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on EP

Source	DF	SS	MS	F	p
cofit	3	0.472	0.157	0.62	0.602
Error	92	20.477	0.253		
Total	94	20.950			

Level	N	Mean	StDev
CPS	43	0.6667	0.6004
PGE	16	0.7982	0.4281
UND	16	0.7654	0.3799
CONT	20	0.8483	0.4084

Pooled StDev = 0.5028



CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control

WOMEN

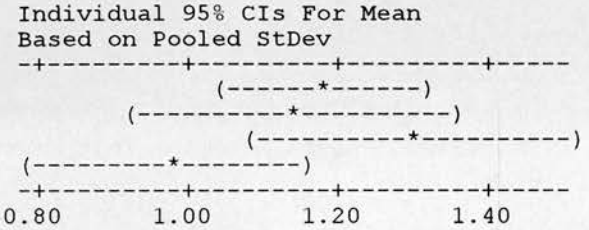
Figure 90:ANOVA OF SI (SEXUAL INHIBITION) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on SI

Source	DF	SS	MS	F	p
FITS	3	1.132	0.377	1.82	0.149
Error	93	20.104	0.207		
Total	94	21.236			

Level	N	Mean	StDev
CPS	43	1.1740	0.4763
PGE	16	1.1372	0.4825
UND	16	1.3006	0.4546
CONT	20	0.9757	0.3891

Pooled StDev = 0.4553



Fisher's pairwise comparisons

Family error rate = 0.201
Individual error rate = 0.0500

Critical value = 1.985

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.2178 0.2914		
3	-0.3811 0.1281	-0.4646 0.1379	
4	-0.0360 0.4328	-0.1228 0.4460	0.0405 0.6093

CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control

WOMEN

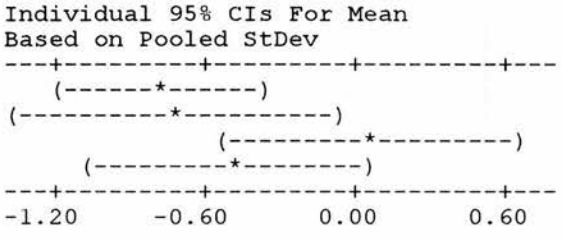
Figure 91:ANOVA OF LF (LENGTH OF FOREPLAY) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on LF

Source	DF	SS	MS	F	p
FITS	3	9.02	3.01	1.76	0.159
Error	96	158.46	1.70		
Total	97	167.48			

Level	N	Mean	StDev
CPS	43	-0.761	1.349
PGE	16	-0.726	1.221
UND	16	0.044	1.322
CONT	20	-0.505	1.265

Pooled StDev = 1.305



CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control

WOMEN

Figure 92:ANOVA OF OE (ORGASM ADEQUACY DURING INTERCOURSE AND ITS EVALUATION) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

Analysis of Variance on OE

```
Source      DF        SS        MS        F        p
FITS         3     3.429     1.143     2.35    0.078
Error       93    39.870     0.486
Total       94    43.299

                                      Individual 95% CIs For Mean
                                      Based on Pooled StDev
 Level       N      Mean     StDev  ----------+---------+---------+------
 CPS        43    0.2159    0.7982            (----*-----)
 PGE        16    0.3186    0.7834            (-------*-------)
 UND        16    0.5963    0.5213                    (--------*--------)
 CONT       20   -0.0511    0.4588   (-------*-------)
                                      ----------+---------+---------+------
Pooled StDev =    0.6973                    0.00      0.40      0.80

Fisher's pairwise comparisons

    Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.989

Intervals for (column level mean) - (row level mean)

               1         2         3

     2   -0.5092
          0.3036

     3   -0.8156   -0.7782
          0.0548    0.2229

     4   -0.1316   -0.0993    0.1532
          0.6655    0.8388    1.1416


CPS = Complex Partial Seizures
PGE = Primary General Epilepsy
UND = Undefined
CONT = Control
```

WOMEN

Figure 93:ANOVA OF MS(MARITAL SATISFACTION) SUBSCALE OF SES 4 FOR SEIZURE TYPE GROUPS.

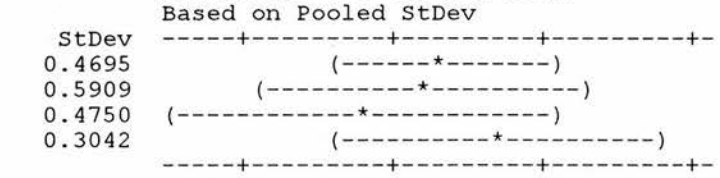
Analysis of Variance on MS4

Source	DF	SS	MS	F	p
cofit	3	0.268	0.089	0.40	0.752
Error	85	18.940	0.223		
Total	88	19.209			

Level	N	Mean	StDev
CPS	43	-0.7305	0.4695
PGE	20	-0.7642	0.5909
UND	15	-0.8377	0.4750
CONT	20	-0.6550	0.3042

Pooled StDev = 0.4720

Individual 95% CIs For Mean Based on Pooled StDev



CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERAL EPILEPSY
UND = UNDEFINED
CONT = CONTROLS

WOMEN

Figure 94:ANOVA OF AE(ATTITUDE TO EXTRAMARITAL INVOLVMENT) SUBSCALE OF SES 4 FOR SEIZURE TYPE

```
Analysis of Variance on AE
Source      DF         SS          MS          F          p
cofit        3      4.254       1.418       2.35      0.078
Error       84     50.634       0.603
Total       87     54.888

                                          Individual 95% CIs For Mean
                                          Based on Pooled StDev
 Level       N       Mean      StDev  ---+---------+---------+---------+---
 CPS        43    -0.3432     0.8337            (-----*------)
 PGE        20    -0.3989     0.7824         (--------*--------)
 UND        15    -0.4523     0.9331      (----------*---------)
 CONT       20     0.1583     0.4506                        (--------*--------)
                                          ---+---------+---------+---------+---
Pooled StDev =    0.7764                 -0.80     -0.40     -0.00      0.40

Fisher's pairwise comparisons

    Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.989

Intervals for (column level mean) - (row level mean)

               1          2          3

     2    -0.3781
           0.4897

     3    -0.3870    -0.5025
           0.6053     0.6092

     4    -0.9433    -1.0652    -1.1727
          -0.0596    -0.0494    -0.0486


Kruskal-Wallis Test


   95 cases were used

LEVEL     NOBS     MEDIAN  AVE. RANK    Z VALUE
    1       43  -0.33000        41.9       -0.82
    2       16  -0.55000        39.4       -0.98
    3       16  -0.08000        40.0       -0.69
    4       20   0.23000        58.6        2.62
OVERALL     95                  44.5

H = 6.99  d.f. = 3  p = 0.073
H = 7.00  d.f. = 3  p = 0.073 (adjusted for ties)


CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERAL EPILEPSY
UND = UNDEFINED
CONT = CONTROLS
```

WOMEN

Figure 95:ANOVA OF ES(EVALUATION OF PARTNER AS SEXUAL PARTNER) SUBSCALE OF SES 4 FOR SEIZURE TYPE

Analysis of Variance on ES

Source	DF	SS	MS	F	p
cofit	3	10.81	3.60	2.32	0.081
Error	93	130.53	1.55		
Total	94	141.34			

Level	N	Mean	StDev
CPS	43	-0.621	1.159
PGE	20	-1.118	1.487
UND	15	-1.431	1.416
CONT	20	-0.427	1.000

Pooled StDev = 1.247

Individual 95% CIs For Mean Based on Pooled StDev

```
-+---------+---------+---------+-----
                 (-----*-----)
        (-------*-------)
 (---------*--------)
                  (-------*-------)
-+---------+---------+---------+-----
-2.10     -1.40     -0.70     -0.00
```

Fisher's pairwise comparisons

Family error rate = 0.200
Individual error rate = 0.0500

Critical value = 1.989

Intervals for (column level mean) - (row level mean)

	1	2	3
2	-0.200 1.193		
3	0.013 1.606	-0.580 1.205	
4	-0.904 0.515	-1.507 0.124	-1.907 -0.102

CPS = COMPLEX PARTIAL SEIZURES
PGE = PRIMARY GENERAL EPILEPSY
UND = UNDEFINED
CONT = CONTROLS

WOMEN

Figure 96:MULTIPLE REGRESSION FOR SES 1 (UNWEIGHTED) SCORES

The regression equation is

SES1 = 54.3 + 0.205 age + 3.00 affiliation - 3.54 FURTHER EDUCATION - 1.22 MARRIED?

243 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	54.280	3.884	13.97	0.000
age	0.2046	0.1312	1.56	0.120
affil	3.002	2.130	1.41	0.160
FURTHED	-3.540	2.055	-1.72	0.086
MARRIED?	-1.218	2.256	-0.54	0.590

s = 15.50 R-sq = 3.3% R-sq(adj) = 1.7%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	1923.2	480.8	2.00	0.095
Error	231	55515.3	240.3		
Total	235	57438.6			

SOURCE	DF	SEQ SS
age	1	769.4
affil	1	375.4
FURTHED	1	708.4
MARRIED?	1	70.1

Figure 97:MULTIPLE REGRESSION FOR SES 2 (UNWEIGHTED) SCORES

The regression equation is

SES2 = 46.7 + 0.0861 age - 0.14 affiliation - 1.21 FURTHER EDUCATION - 0.16 MARRIED?

243 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	46.671	2.232	20.91	0.000
age	0.08607	0.07542	1.14	0.255
affil	-0.142	1.224	-0.12	0.908
FURTHED	-1.207	1.181	-1.02	0.308
MARRIED?	-0.163	1.297	-0.13	0.900

s = 8.910 R-sq = 1.1% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	209.16	52.29	0.66	0.621
Error	242	18337.82	79.38		
Total	243	18546.98			

SOURCE	DF	SEQ SS
age	1	121.88
affil	1	3.38
FURTHED	1	82.65
MARRIED?	1	1.25

WOMEN

Figure 98:MULTIPLE REGRESSION FOR SES 3 (UNWEIGHTED) SCORES

The regression equation is

SES3 = 56.0 + 0.399 age - 1.32 affiliation - 9.96 FURTHER EDUCATION

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	56.020	9.070	6.18	0.000
age	0.3992	0.2488	1.60	0.112
affil	-1.323	4.479	-0.30	0.768
FURTHED	-9.958	4.135	-2.41	0.018

s = 19.73 R-sq = 8.8% R-sq(adj) = 5.8%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	3419.9	1140.0	2.93	0.038
Error	93	35415.5	389.2		
Total	94	38835.3			

SOURCE	DF	SEQ SS
age	1	1015.5
affil	1	147.4
FURTHED	1	2257.1

Figure 99:MULTIPLE REGRESSION FOR SES 3 (UNWEIGHTED) SCORES

The regression equation is

SES4 = 74.7 + 0.065 age - 4.19 FURTHED + 0.98 affil

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	74.654	5.509	13.55	0.000
age	0.0647	0.1264	0.51	0.610
FURTHED	-4.186	2.086	-2.01	0.048
affil	0.984	2.642	0.37	0.711

s = 9.736 R-sq = 5.1% R-sq(adj) = 1.9%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	449.88	149.96	1.58	0.199
Error	88	8341.20	94.79		
Total	91	8791.08			

SOURCE	DF	SEQ SS
age	1	31.35
FURTHED	1	405.39
affil	1	13.14

F-to-Enter: 4.00 F-to-Remove: 4.00

Multiple Regression

Response is SES4 on 3 predictors, with N = 95

Step	1
Constant	77.17
FURTHED	-4.3
T-Ratio	-2.10
S	9.65
R-Sq	4.65

WOMEN

Figure 100:MULTIPLE REGRESSION FOR PS (PREMARITAL SEXUAL EXPERIENCE) SUBSCALE OF SES 1

The regression equation is

PS = - 0.631 + 0.00924 age + 0.244 affiliation + 0.0242 FURTHER EDUCATION + 0.010 MARRIED?

```
243 cases used
Predictor       Coef        Stdev    t-ratio        p
Constant     -0.6307       0.1858      -3.40    0.001
age         0.009237     0.006279       1.47    0.143
affil         0.2436       0.1012       2.41    0.017
FURTHED      0.02424      0.09794       0.25    0.805
MARRIED?      0.0104       0.1077       0.10    0.923

s = 0.7336      R-sq = 4.3%      R-sq(adj) = 2.6%
```

Analysis of Variance

```
SOURCE       DF          SS          MS          F        p
Regression    4      5.4492      1.3623       2.53    0.041
Error       228    122.6968      0.5381
Total       232    128.1460

SOURCE       DF      SEQ SS
age           1      2.1967
affil         1      3.2144
FURTHED       1      0.0330
MARRIED?      1      0.0051

F-to-Enter:      4.00    F-to-Remove:      4.00

 Response is    PS    on  3 predictors, with N =  244

    Step        1
Constant  -0.2867

affil       0.65
T-Ratio     5.09

S          0.711
R-Sq        9.99
```

WOMEN

Figure 101:MULTIPLE REGRESSION FOR MS (SEXUAL MORALITY WITHIN MARRIAGE) SUBSCALE OF SES 1

The regression equation is

MS = - 0.227 + 0.0106 age + 0.0877 affiliation - 0.150 FURTHER EDUCATION - 0.0483 MARRIED?

243 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.2267	0.1466	-1.55	0.123
age	0.010584	0.004954	2.14	0.055
affil	0.08771	0.07988	1.10	0.273
FURTHED	-0.15020	0.07728	-1.94	0.053
MARRIED?	-0.04834	0.08496	-0.57	0.570

s = 0.5788 R-sq = 4.5% R-sq(adj) = 2.8%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	3.5946	0.8987	2.68	0.032
Error	228	76.3901	0.3350		
Total	232	79.9847			

SOURCE	DF	SEQ SS
age	1	1.9434
affil	1	0.2766
FURTHED	1	1.2662
MARRIED?	1	0.1085

WOMEN

```
F-to-Enter:       4.00    F-to-Remove:       4.00

 Response is    MS    on  4 predictors, with N =  233
 N(cases with missing obs.) =  11 N(all cases) =  244

    Step         1         2
Constant-0.006111  0.070784

affil         0.40      0.43
T-Ratio       3.81      4.16

FURTHED                -0.195
T-Ratio                 -2.59

S            0.571     0.564
R-Sq          5.92      8.58
```

WOMEN

Figure 102:MULTIPLE REGRESSION FOR SS (SEXUAL SOCIALISATION) SUBSCALE OF SES 1

The regression equation is

SS = - 0.016 + 0.00104 age - 0.0222 affiliation - 0.182 FURTHER EDUCATION - 0.0346 MARRIED?

```
243 cases used

Predictor       Coef        Stdev    t-ratio        p
Constant     -0.0158       0.1610      -0.10    0.922
age         0.001040     0.005437       0.19    0.848
affil       -0.02222      0.08787      -0.25    0.801
FURTHED     -0.18221      0.08490      -2.15    0.033
MARRIED?    -0.03456      0.09407      -0.37    0.714

s = 0.6326      R-sq = 2.2%      R-sq(adj) = 0.4%
```

Analysis of Variance

```
SOURCE       DF          SS          MS          F        p
Regression    4      2.0035      0.5009       1.25    0.290
Error       225     90.0515      0.4002
Total       229     92.0550

SOURCE       DF      SEQ SS
age           1      0.0098
affil         1      0.0851
FURTHED       1      1.8546
MARRIED?      1      0.0540

 Response is    SS    on  4 predictors, with N =  244

    Step         1
Constant  -0.01174

FURTHED     -0.185
T-Ratio      -2.20

S            0.629
R-Sq          2.09
```

Figure 103:MULTIPLE REGRESSION FOR AE (AROUSAL BY EROTIC IMAGERY) SUBSCALE OF SES2

The regression equation is

AE = - 0.167 + 0.00579 age - 0.136 affiliation - 0.125 FURTHER EDUCATION - 0.123 MARRIED?

```
243 cases used

Predictor       Coef        Stdev    t-ratio        p
Constant     -0.1673       0.2742      -0.61    0.542
age         0.005789     0.009210       0.63    0.530
affil        -0.1359       0.1475      -0.92    0.358
FURTHED      -0.1255       0.1438      -0.87    0.384
MARRIED?     -0.1233       0.1565      -0.79    0.432

s = 1.069       R-sq = 1.1%      R-sq(adj) = 0.0%
```

WOMEN

Figure 104:MULTIPLE REGRESSION FOR IS (INTERPERSONAL SEXUAL ATTRACTION) SUBSCALE OF SES2

The regression equation is
IS = - 0.598 + 0.00788 age + 0.445 affil + 0.075 MARRIED? - 0.217 FURTHED

243 CASES

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.5977	0.1893	-3.16	0.002
age	0.007885	0.006524	1.21	0.228
affil	0.4446	0.1431	3.11	0.002
MARRIED?	0.0751	0.1119	0.67	0.503
FURTHED	-0.2171	0.1025	-2.12	0.035

s = 0.7547 R-sq = 7.3% R-sq(adj) = 5.6%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	9.9867	2.4967	4.38	0.002
Error	224	127.5859	0.5696		
Total	228	137.5726			

SOURCE	DF	SEQ SS
age	1	2.7067
affil	1	4.4901
MARRIED?	1	0.2332
FURTHED	1	2.5568

Response is IS on 4 predictors, with N = 244

Step	1	2
Constant	-0.4277	-0.3383
affil	0.43	0.48
T-Ratio	3.03	3.35
FURTHED		-0.23
T-Ratio		-2.24
S	0.763	0.757
R-Sq	3.89	5.98

Figure 105:MULTIPLE REGRESSION FOR EE (EVALUATION OF EXPOSURE TO EROTIC IMAGERY) SUBSCALE OF SES2

The regression equation is

EE = - 0.019 + 0.00672 age - 0.056 affiliation - 0.025 FURTHER EDUCATION- 0.037 MARRIED?

243 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.0194	0.2065	-0.09	0.925
age	0.006725	0.006985	0.96	0.337
affil	-0.0558	0.1128	-0.49	0.621
FURTHED	-0.0246	0.1091	-0.23	0.822
MARRIED?	-0.0365	0.1212	-0.30	0.763

s = 0.8111 R-sq = 0.5% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	4	0.7745	0.1936	0.29	0.881
Error	242	147.3629	0.6579		
Total	243	148.1373			

SOURCE	DF	SEQ SS
age	1	0.4972
affil	1	0.1832
FURTHED	1	0.0343
MARRIED?	1	0.0597

WOMEN

Figure 106:MULTIPLE REGRESSION FOR FI (PREFERRED FREQUENCY OF INTERCOURSE) SUBSCALE OF SES3

The regression equation is

FI = 0.058 + 0.0063 age - 0.033 affiliation + 0.085 FURTHER EDUCATION

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	0.0582	0.5710	0.10	0.919
age	0.00627	0.01173	0.53	0.595
affil	-0.0329	0.1368	-0.24	0.811
FURTHED	0.0849	0.1951	0.44	0.664

s = 0.8614 R-sq = 0.7% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	0.4044	0.1348	0.18	0.909
Error	93	60.8462	0.7420		
Total	94	61.2506			

SOURCE	DF	SEQ SS
age	1	0.2374
affil	1	0.0263
FURTHED	1	0.1407

Figure 107:MULTIPLE REGRESSION FOR EP (ENJOYMENT POTENTIAL) SUBSCALE OF SES3

The regression equation is

EP = 1.02 + 0.00365 age - 0.0261 affiliation - 0.204 FURTHER EDUCATION

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	1.0177	0.3313	3.07	0.003
age	0.003650	0.006809	0.54	0.593
affil	-0.02615	0.07979	-0.33	0.744
FURTHED	-0.2036	0.1140	-1.79	0.078

s = 0.4971 R-sq = 4.5% R-sq(adj) = 0.9%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	0.9392	0.3131	1.27	0.291
Error	93	20.0173	0.2471		
Total	94	20.9564			

SOURCE	DF	SEQ SS
age	1	0.0652
affil	1	0.0858
FURTHED	1	0.7881

WOMEN

Figure 108:MULTIPLE REGRESSION FOR SI (SEXUAL INHIBITION) SUBSCALE OF SES3

The regression equation is

SI = 1.04 + 0.00200 age + 0.147 affiliation - 0.140 FURTHER EDUCATION

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	1.0414	0.2275	4.58	0.000
age	0.001995	0.006297	0.32	0.752
affil	0.1474	0.1068	1.38	0.171
FURTHED	-0.1400	0.1023	-1.37	0.175

s = 0.4631 R-sq = 4.1% R-sq(adj) = 0.7%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	0.7703	0.2568	1.20	0.316
Error	93	17.8009	0.2145		
Total	94	18.5712			

SOURCE	DF	SEQ SS
age	1	0.0332
affil	1	0.3357
FURTHED	1	0.4014

Figure 109:MULTIPLE REGRESSION FOR LF (LENGTH OF FOREPLAY) SUBSCALE OF SES3

The regression equation is
LF = - 0.870 + 0.0112 age - 0.352 affil + 0.022 FURTHED

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.8698	0.8019	-1.08	0.281
age	0.01119	0.01867	0.60	0.550
affil	-0.3518	0.3993	-0.88	0.381
FURTHED	0.0221	0.3021	0.07	0.942

s = 1.348 R-sq = 1.4% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	2.075	0.692	0.38	0.767
Error	92	147.207	1.817		
Total	94	149.283			

SOURCE	DF	SEQ SS
age	1	0.583
affil	1	1.482
FURTHED	1	0.010

Figure 110:MULTIPLE REGRESSION FOR OE (ORGASM ADEQUACY DURING INTERCOURSE AND ITS EVALUATION) SUBSCALE OF SES 3 FOR SEIZURE TYPE GROUPS

The regression equation is

OE = 0.211 - 0.00096 age + 0.003 affiliation + 0.059 FURTHER EDUCATION

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	0.2110	0.3448	0.61	0.542
age	-0.000957	0.009665	-0.10	0.921
affil	0.0026	0.1598	0.02	0.987
FURTHED	0.0589	0.1521	0.39	0.700

s = 0.6631 R-sq = 0.2% R-sq(adj) = 0.0%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	0.0735	0.0245	0.06	0.983
Error	93	33.8580	0.4397		
Total	94	33.9315			

SOURCE	DF	SEQ SS
age	1	0.0072
affil	1	0.0004
FURTHED	1	0.0659

Figure 111:MULTIPLE REGRESSION FOR MS (MARITAL SATISFACTION) SUBSCALE OF SES 4

The regression equation is

MS = - 0.117 - 0.0119 age - 0.145 FURTHED + 0.023 affil

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	-0.1169	0.2628	-0.44	0.658
age	-0.011911	0.006103	-1.95	0.054
FURTHED	-0.1454	0.1007	-1.44	0.152
affil	0.0234	0.1320	0.18	0.860

s = 0.4614 R-sq = 6.7% R-sq(adj) = 3.4%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	1.2825	0.4275	2.01	0.119
Error	93	17.8825	0.2129		
Total	94	19.1650			

SOURCE	DF	SEQ SS
age	1	0.8130
FURTHED	1	0.4628
affil	1	0.0067

WOMEN

Figure 112:MULTIPLE REGRESSION FOR AE (ATTITUDE TO EXTRAMARITAL INVOLVEMENT) SUBSCALE OF SES 4

The regression equation is
AE = 0.898 - 0.0164 age - 0.381 FURTHED - 0.057 affil

```
95 cases used
Predictor       Coef       Stdev    t-ratio        p
Constant      0.8982      0.4423       2.03    0.046
age         -0.01642     0.01030      -1.60    0.114
FURTHED       -0.3814      0.1701      -2.24    0.028
affil         -0.0575      0.2222      -0.26    0.797

s = 0.7765      R-sq = 8.5%      R-sq(adj) = 5.2%

Analysis of Variance

SOURCE       DF          SS          MS         F        p
Regression    3      4.6631      1.5544      2.58    0.059
Error        93     50.0452      0.6030
Total        94     54.7083

SOURCE       DF       SEQ SS
age           1       1.6325
FURTHED       1       2.9903
affil         1       0.0403

R denotes an obs. with a large st. resid.
```

Stepwise Regression

```
 F-to-Enter:       4.00    F-to-Remove:       4.00

 Response is   AE    on  3 predictors, with N =   95


    Step         1
Constant    0.3390

FURTHED      -0.38
T-Ratio      -2.24

S            0.780
R-Sq          5.57
```

WOMEN

Figure 113:MULTIPLE REGRESSION FOR ES(EVALUATION OF PARTNER AS SEXUAL PARTNER) SUBSCALE OF SES 4

The regression equation is

ES = 0.983 - 0.0446 age - 0.236 FURTHED + 0.358 affil

95 cases used

Predictor	Coef	Stdev	t-ratio	p
Constant	0.9827	0.7048	1.39	0.167
age	-0.04456	0.01640	-2.72	0.008
FURTHED	-0.2360	0.2710	-0.87	0.386
affil	0.3582	0.3541	1.01	0.315

s = 1.237 R-sq = 9.9% R-sq(adj) = 6.7%

Analysis of Variance

SOURCE	DF	SS	MS	F	p
Regression	3	13.969	4.656	3.04	0.033
Error	93	127.035	1.531		
Total	94	141.004			

SOURCE	DF	SEQ SS
age	1	10.902
FURTHED	1	1.501
affil	1	1.566

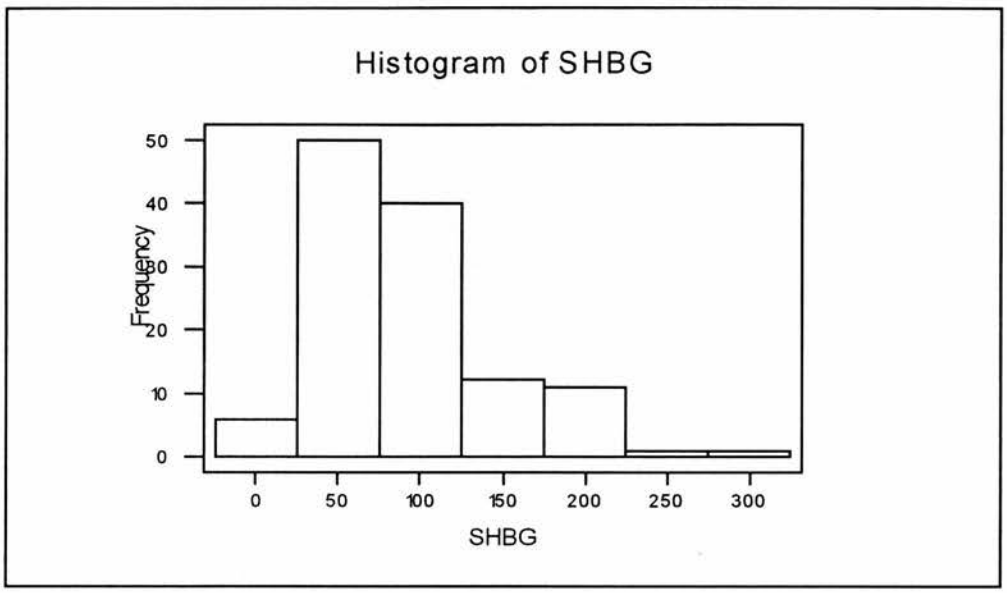
Stepwise Regression

Response is ES4 on 3 predictors, with N = 95

Step	1
Constant	0.6404
age	-0.044
T-Ratio	-2.67
S	1.24
R-Sq	7.73

WOMEN

Figure 114:ANOVA OF SEX HORMONE BINDING GLOBULIN FOR UNTREATED PROLIFERATIVE,MIDCYCLE AND LUTEAL PHASES




```
Analysis of Variance on SHBG
Source     DF        SS        MS        F        p
C27         2      5898      2949     3.04    0.059
Error      39     37876       971
Total      42     43774
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  -------+---------+---------+---------
 SHBGp     18     50.44     14.88       (------*-------)
 SHBGl     14     74.50     44.68                 (-------*--------)
 SHBGm     11     47.70     30.12    (---------*---------)
                                    -------+---------+---------+---------
Pooled StDev =    31.16                40        60        80
```

Fisher's pairwise comparisons

Family error rate = 0.120

Individual error rate = 0.0500

Critical value = 2.023

Intervals for (column level mean) - (row level mean)

```
            1          2

    2    -46.52

•          1.59

    3    -22.12       0.70
          27.61      52.90
```

SHBGp Proliferative phase SHBG
SHBGl Luteal phase SHBG
SHBGm Midcycle phase SHBG

WOMEN

SHBG Values skewed. Not improved with logarithmic transformation.
Comparisons checked using Kruskal-Wallis

43 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	18	46.00	20.0	-0.67
2	14	65.50	27.1	2.08
3	11	43.50	16.4	-1.52
OVERALL	43		21.5	

H = 4.91 d.f. = 2 p = 0.086
H = 4.91 d.f. = 2 p = 0.086 (adjusted for ties)

Figure 115:ANOVA OF SEX HORMONE BINDING GLOBULIN FOR TREATED PROLIFERATIVE,MIDCYCLE AND LUTEAL PHASES

```
Analysis of Variance on SHBG(treated)
Source     DF        SS        MS        F        p
C37         2     13856      6928     2.32    0.105
Error      86    226619      2982
Total      87    240476
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  --+---------+---------+---------+----
 SHBGp     27    122.26     48.97              (--------*--------)
 SHBGl     31    112.89     63.48           (-------*--------)
 SHBGm     30     90.83     49.68  (-------*-------)
                                  --+---------+---------+---------+----
Pooled StDev =    54.61             75        100       125       150
```

Fisher's pairwise comparisons

Family error rate = 0.121

Individual error rate = 0.0500

Critical value = 1.992

Intervals for (column level mean) - (row level mean)

	1	2
2	-21.5 40.2	
3	1.1 61.8	-7.0 51.2

Kruskal-Wallis

88 cases were used

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
SHBGp	27	114.00	47.4	1.85
SHBGl	30	104.00	40.7	0.21
SHBGm	31	94.00	33.4	-1.94
OVERALL	88		40.0	

H = 4.83 d.f. = 2 p = 0.090
H = 4.84 d.f. = 2 p = 0.090 (adjusted for ties)

Figure 116:TWOSAMPLE T TEST COMPARING TREATED AND UNTREATED PROLIFERATIVE PHASE SHBG

Twosample T for UNTREATED vs TREATED

	N	Mean	StDev	SE Mean
UNTREATED	18	50.4	14.9	3.5
TREATED	27	122.3	49.0	10

95% C.I. for mu C48 - mu C49: (-94.0, -50)
T-Test mu C48 = mu C49 (vs not =): T= -6.65 **P=0.0000** DF= 27

Figure 117:TWOSAMPLE T TEST COMPARING TREATED AND UNTREATED LUTEAL PHASE SHBG

Twosample T for UNTREATED vs TREATED

	N	Mean	StDev	SE Mean
UNTREATED	14	74.5	44.7	12
TREATED	31	112.9	63.5	12

95% C.I. for mu C48 - mu C49: (-73, -4)
T-Test mu C48 = mu C49 (vs not =): T= -2.25 **P=0.031** DF= 35

Figure 118:TWOSAMPLE T TEST COMPARING TREATED AND UNTREATED MIDCYCLE PHASE SHBG

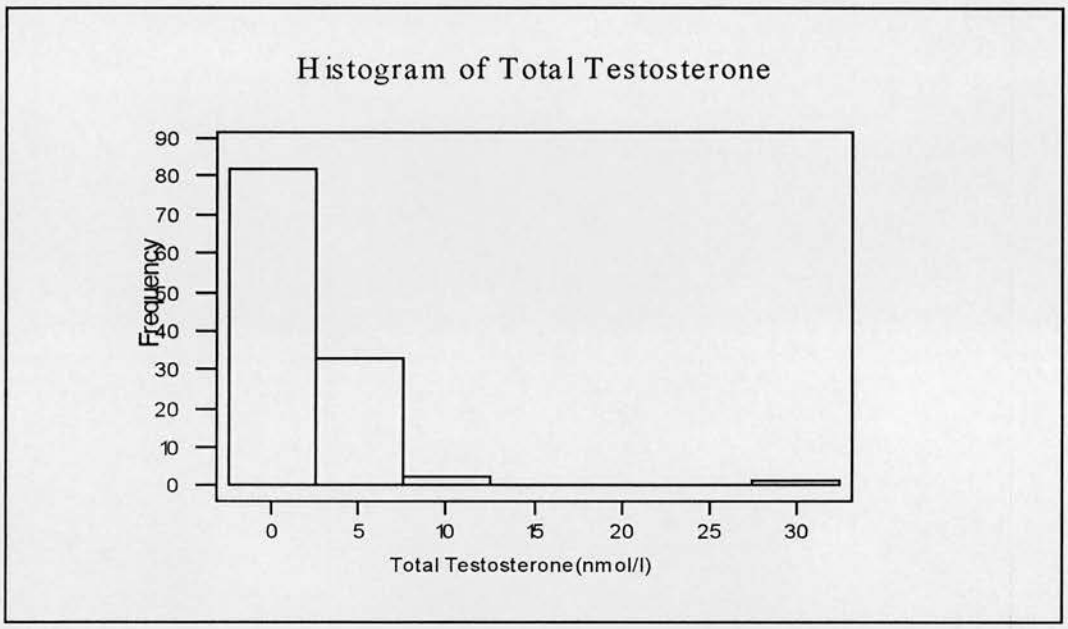
Twosample T for UNTREATED vs TREATED

	N	Mean	StDev	SE Mean
Treated	11	79.8	49.0	7.8
Untreated	30	47.7	30.1	9.5

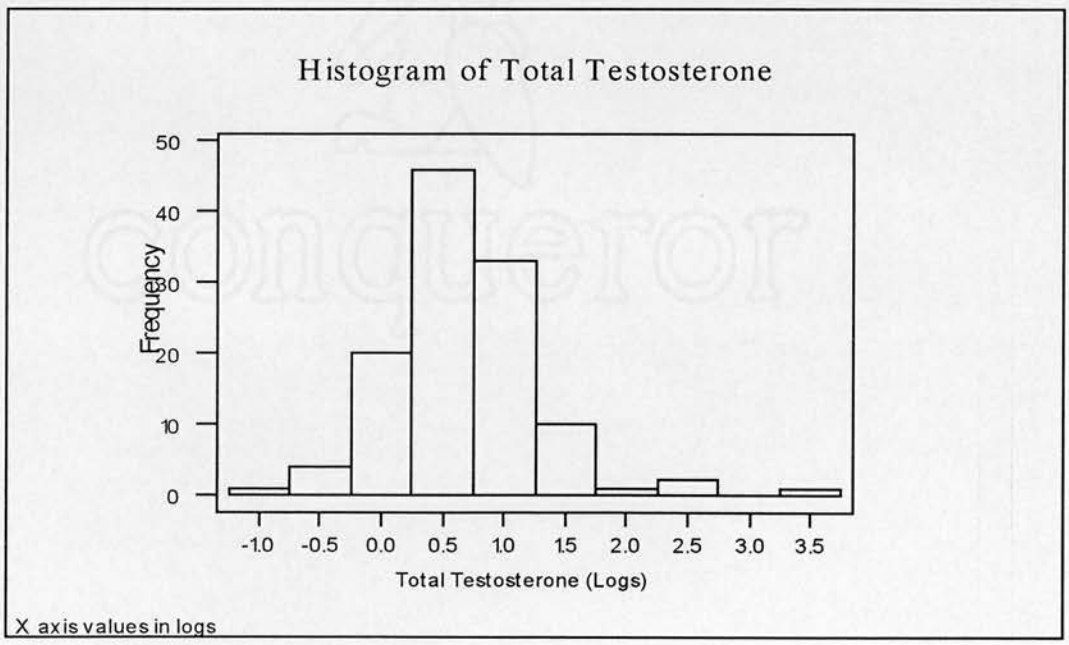
95% C.I. for mu C55 - mu C56: (6.5, 57.7)
T-Test mu C55 = mu C56 (vs not =): T= 2.60 **P=0.016** DF= 22

WOMEN

Figure 119:ANOVA OF TOTAL TESTOSTERONE (UNTREATED)



Because values not normally distributed values transformed into logs



WOMEN

Figure 120:ANALYSIS OF VARIANCE OF TOTAL TESTOSTERONE FOR UNTREATED PROLIFERATIVE, MIDCYCLE AND LUTEAL GROUPS

Analysis of Variance on Total testosterone (Logs)

Source	DF	SS	MS	F	p
group	2	0.554	0.277	1.39	0.262
Error	40	7.791	0.200		
Total	42	8.344			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	--------+---------+---------+--------
Prolif	18	0.2534	0.4675	(----------*---------)
luteal	14	0.5184	0.3307	(----------*----------)
midcycle	11	0.3795	0.5429	(-------------*-------------)
				--------+---------+---------+--------
Pooled StDev =		0.4469		0.20 0.40 0.60

Fisher's pairwise comparisons

Family error rate = 0.120

Individual error rate = 0.0500

Critical value = 2.023

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.5872 0.0572	
3	-0.4827 0.2306	-0.2354 0.5133

WOMEN

Figure 121:ANOVA OF TOTAL TESTOSTERONE USING NORMAL VALUES (nmol/L)

```
Analysis of Variance on Total Testosterone (Nmol/L)
Source      DF         SS         MS          F          p
group        2      1.092      0.546       1.04      0.362
Error       40     20.439      0.524
Total       42     21.531
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level       N       Mean      StDev  -----+---------+---------+---------+-
  Prolif    18     1.4111     0.5645   (-----------*-----------)
  Luteal    14     1.7571     0.5049              (------------*------------)
  Midcycle  11     1.7025     1.1405         (---------------*--------------)
                                       -----+---------+---------+---------+-
Pooled StDev =    0.7239               1.20      1.50      1.80      2.10

Fisher's pairwise comparisons
   Family error rate = 0.120
Individual error rate = 0.0500

Critical value = 2.023
Intervals for (column level mean) - (row level mean)

             1         2


     2    -0.8679
           0.1758

     3    -0.8690   -0.5518
           0.2862    0.6610
```

WOMEN

Figure 122:ANALYSIS OF VARIANCE OF TOTAL TESTOSTERONE FOR TREATED PROLIFERATIVE, MIDCYCLE AND LUTEAL GROUPS

Analysis of Variance on Total Testosterone (treated) values in nmol/l

Source	DF	SS	MS	F	p
GROUP	2	12.9	6.5	0.43	0.653
Error	86	1099.0	15.1		
Total	87	1112.0			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	--+---------+---------+---------+----
Prolif	27	3.526	1.267	(----------*----------)
Luteal	31	2.511	1.914	(----------*----------)
Midcycle	30	3.067	2.364	(----------*----------)
				--+---------+---------+---------+----
Pooled StDev =		3.880		1.2 2.4 3.6 4.8

Fisher's pairwise comparisons

Family error rate = 0.121

Individual error rate = 0.0500

Critical value = 1.993

Intervals for (column level mean) - (row level mean)

	1	*2*
2	-1.179 3.209	
3	-1.754 2.673	-2.680 1.569

Figure 123:ANOVA OF TOTAL TESTOSTERONE (VALUED IN LOGS)

Analysis of Variance on Total Testosterone (logs)

Source	DF	SS	MS	F	p
Group	2	0.286	0.143	0.36	0.699
Error	84	29.046	0.398		
Total	87	29.332			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-----+---------+---------+---------+-
Prolif	27	0.8492	0.6869	(----------*----------)
Luteal	31	0.7574	0.5520	(----------*----------)
Midcycle	30	0.9026	0.6555	(----------*----------)
				-----+---------+---------+---------+-
Pooled StDev =		0.6308		0.60 0.80 1.00 1.20

Figure 124:TWOSAMPLE T TEST COMPARING UNTREATED GROUP AND TREATED GROUP PROLIFERATIVE PHASE TOTAL TESTOSTERONE

Twosample T for Untreated TT v Treated TT(Values in Logs)

	N	Mean	StDev	SE Mean
TTu	18	0.253	0.468	0.11
TTtr	27	0.849	0.687	0.14

95% C.I. for mu C110 - mu C111: (-0.96, -0.23)
T-Test mu C110 = mu C111 (vs not =): T= -3.30 **P=0.0021** DF= 38

Figure 125:TWOSAMPLE T TEST COMPARING UNTREATED GROUP AND TREATED GROUP LUTEAL PHASE TOTAL TESTOSTERONE

Twosample T for Untreated v Treated Luteal TT (values in logs)

	N	Mean	StDev	SE Mean
TTu	14	0.518	0.331	0.088
TTtr	31	0.757	0.552	0.11

95% C.I. for mu C112 - mu C113: (-0.519, 0.04)
T-Test mu C112 = mu C113 (vs not =): T= -1.73 P=0.092 DF= 38

Figure 126:TWOSAMPLE T TEST COMPARING UNTREATED GROUP AND TREATED GROUP MIDCYCLE PHASE TOTAL TESTOSTERONE

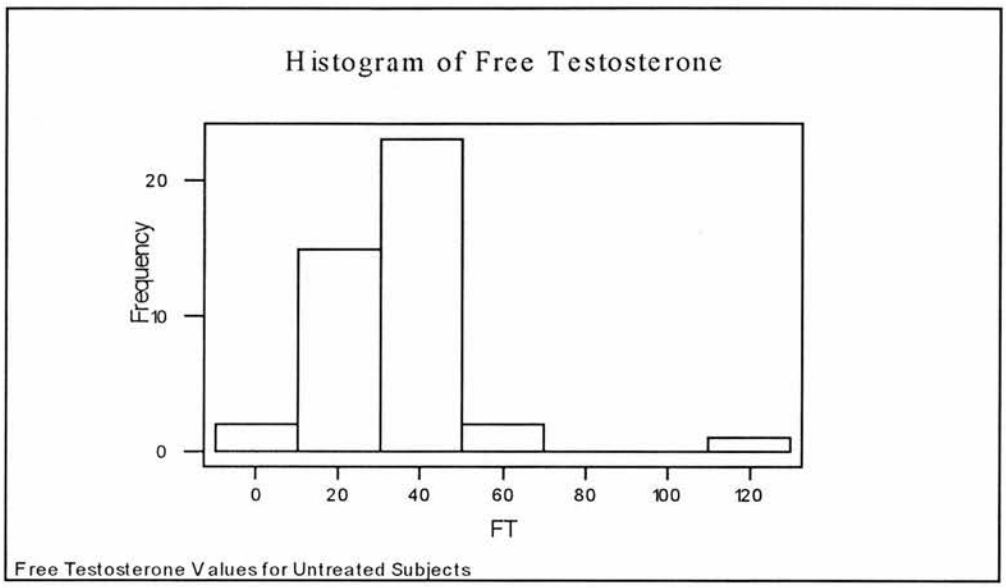
Twosample T for Untreated v Treated midcycle TT (values in logs)

	N	Mean	StDev	SE Mean
TTu	11	0.379	0.543	0.17
TTtr	30	0.903	0.655	0.13

95% C.I. for mu C114 - mu C115: (-0.97, -0.07)
T-Test mu C114 = mu C115 (vs not =): T= -2.44 **P=0.025** DF= 19

TTu Untreated Groups Total testosterone
TTtr Treated Groups Total testosterone

Figure 127:ANALYSIS OF VARIANCE OF CALCULATED FREE TESTOSTERONE FOR UNTREATED PROLIFERATIVE, MIDCYCLE AND LUTEAL GROUPS



Analysis of Variance

Source	DF	SS	MS	F	p
Factor	2	383	191	0.60	0.555
Error	40	12792	320		
Total	42	13175			

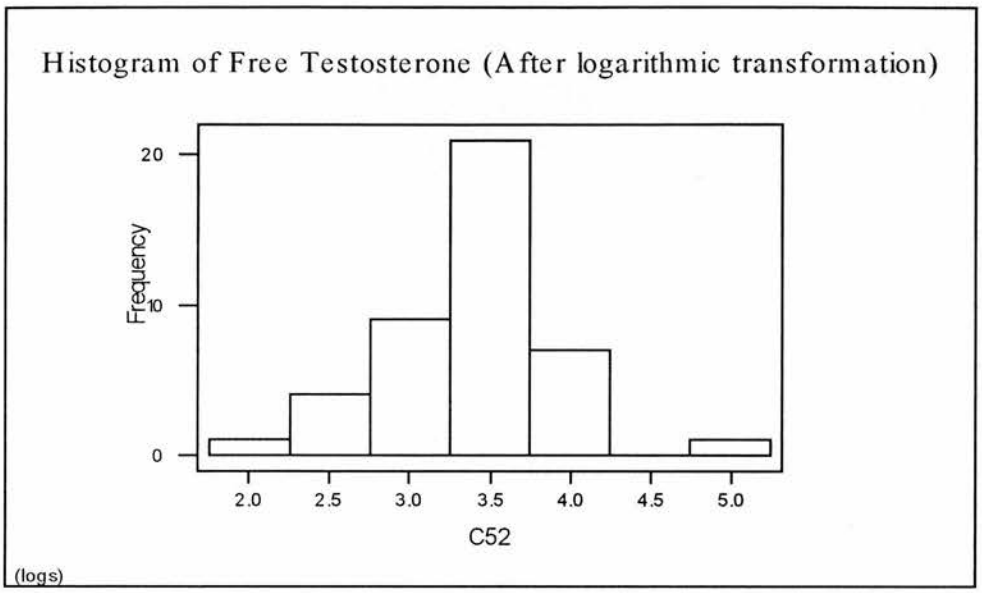
Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	----+---------+---------+---------+--
FTp	18	29.84	12.29	(---------*---------)
FTl	14	33.00	13.17	(----------*----------)
FTm	11	37.54	29.43	(-------------*-------------)
				----+---------+---------+---------+--
Pooled StDev =		17.88		24.0 32.0 40.0 48.0

FTp = Proliferative FT
Ftl = Luteal FT
Ftm = Midcycle FT

Figure 128:ANALYSIS OF VARIANCE OF CALCULATED FREE TESTOSTERONE FOR UNTREATED PROLIFERATIVE, MIDCYCLE AND LUTEAL GROUPS

AFTER LOGARITHMIC TRANSFORMATION



X Axis = Logarithmic values of FT

Analysis of Variance

Source	DF	SS	MS	F	p
Factor	2	0.222	0.111	0.41	0.668
Error	40	10.880	0.272		
Total	42	11.102			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	---------+---------+---------+-------
FTp	18	3.2877	0.5246	(---------*--------)
FTl	14	3.3935	0.5177	(----------*----------)
FTm	11	3.4662	0.5219	(-------------*------------)

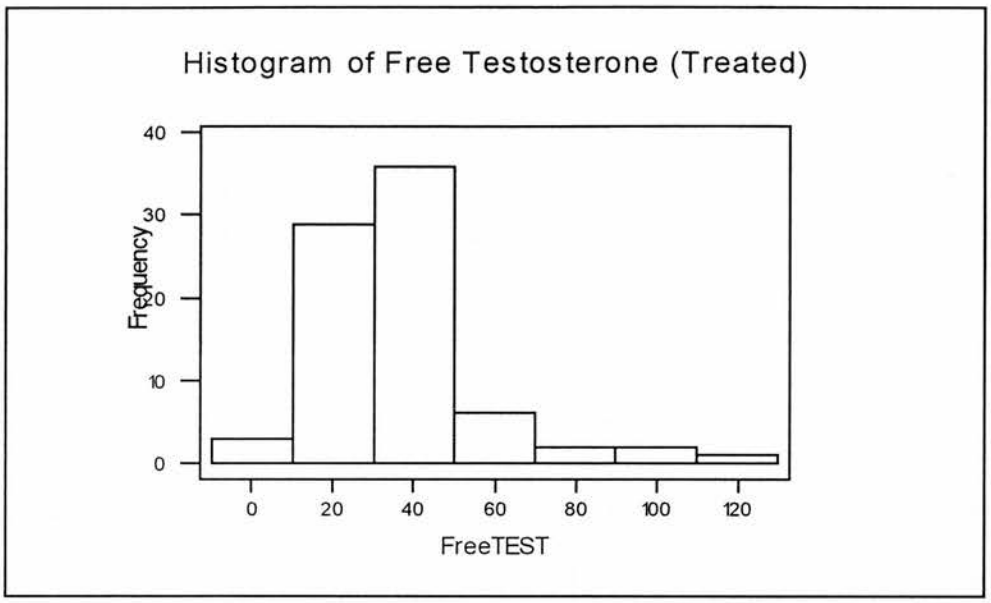
---------+---------+---------+-------

Pooled StDev = 0.5215 3.25 3.50 3.75

FTp = Proliferative FT
Ftl = Luteal FT
Ftm = Midcycle FT

WOMEN

Figure 129:ANALYSIS OF VARIANCE OF CALCULATED FREE TESTOSTERONE FOR TREATED PROLIFERATIVE, MIDCYCLE AND LUTEAL GROUPS




```
Analysis of Variance on FT
Source      DF         SS         MS         F         p
C60          2       2743       1372      3.73     0.028
Error       86      27941        368
Total       87      30685
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level       N       Mean      StDev  ---+---------+---------+---------+---
     FTp    27      29.99      18.47    (--------*---------)
     FTl    31      30.08      15.24     (--------*--------)
     FTm    30      42.46      23.15                       (--------*--------)
                                       ---+---------+---------+---------+---
Pooled StDev =     19.17               24.0       32.0       40.0       48.0
```

Fisher's pairwise comparisons

Family error rate = 0.121

Individual error rate = 0.0500

Critical value = 1.992

Intervals for (column level mean) - (row level mean)

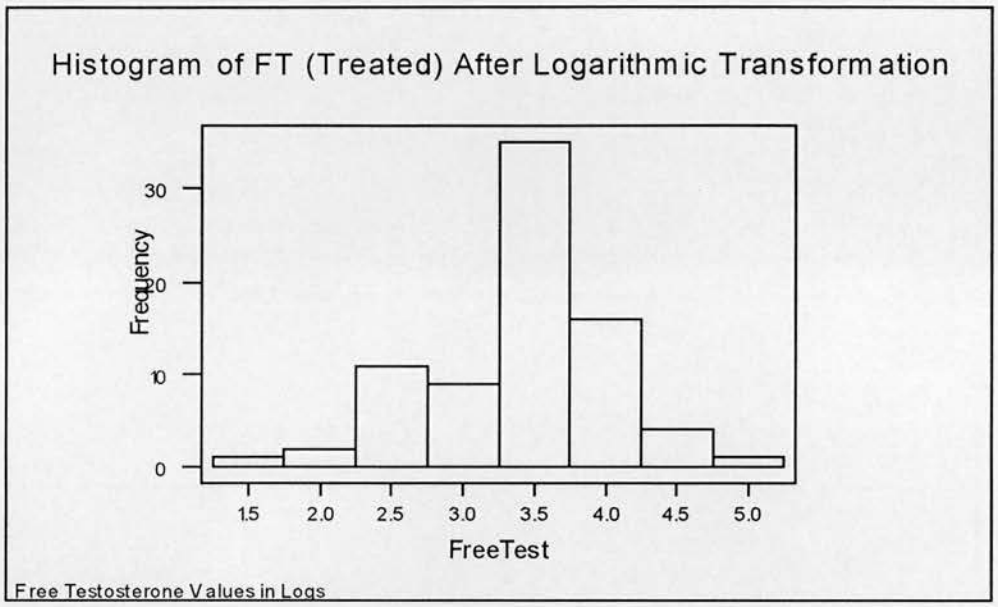
```
            1       2

     2    -10.75
           10.58

     3    -23.31    -22.60
           1.64      -2.17
```

FTp = Proliferative FT
Ftl = Luteal FT
Ftm = Midcycle FT

WOMEN

Figure 130:ANALYSIS OF VARIANCE OF CALCULATED FREE TESTOSTERONE FOR TREATED PROLIFERATIVE, MIDCYCLE AND LUTEAL GROUPS

(AFTER LOGARITHMIC TRANSFORMATION OF FREE TESTOSTERONE)




```
Analysis of Variance on FreeTest(Logs)
Source     DF        SS        MS        F        p
C60         2     2.811     1.406     4.30    0.017
Error      86    24.816     0.327
Total      87    27.627
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  ----------+---------+---------+------
  FTp 1     27    3.2447    0.5688  (---------*--------)
  FTl 2     31    3.2332    0.6552   (-------*--------)
  FTm 3     30    3.6359    0.4674                     (-------*--------)
                                    ----------+---------+---------+------
Pooled StDev =   0.5714                      3.25      3.50      3.75
```

Fisher's pairwise comparisons

```
   Family error rate = 0.121
Individual error rate = 0.0500
```

Critical value = 1.992

```
Intervals for (column level mean) - (row level mean)
            1         2

     2   -0.3064
          0.3293

     3   -0.7142   -0.7071
          0.0682   -0.0983
```

```
FTp = Proliferative FT
Ftl = Luteal FT                Ftm = Midcycle FT
```

Figure 131:TWOSAMPLE T TEST COMPARING UNTREATED GROUP AND TREATED GROUP PROLIFERATIVE PHASE FREE TESTOSTERONE

Twosample T for Untreated v Treated proliferative FT

```
      N      Mean      StDev   SE Mean
FTu  18      29.8       12.3       2.9
FTt  27      30.0       18.5       3.9

95% C.I. for mu C12 - mu C13: ( -9.9,  9.6)
T-Test mu C12 = mu C13 (vs not =): T= -0.03  P=0.97  DF=  38


FTu  Untreated Groups FT
FTt  Treated Groups FT
```

Figure 132:TWOSAMPLE T TEST COMPARING UNTREATED GROUP AND TREATED GROUP MIDCYCLE PHASE FREE TESTOSTERONE

Twosample T for FTu vs FTt

```
      N      Mean      StDev   SE Mean
FTu  11      37.5       29.4       9.3
FTt  30      42.5       23.2       4.5

95% C.I. for mu FTu - mu FTt: ( -27.2,  17.4)
T-Test mu FTu = mu FTt (vs not =): T= -0.48  P=0.64  DF=  13

Ftu  Untreated Groups FT
FTt  Treated Groups FT
```

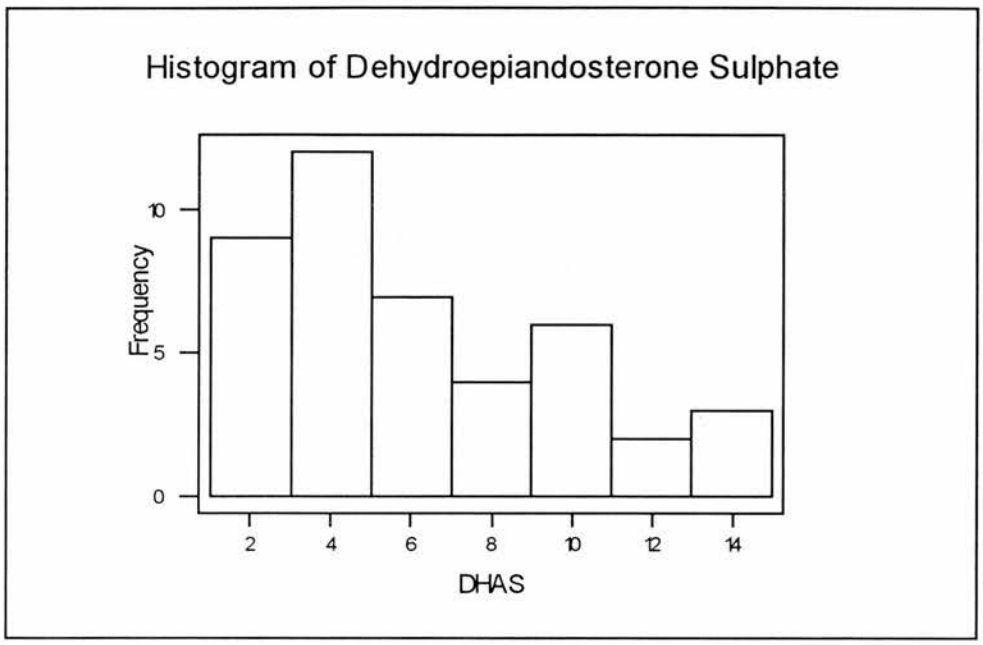
Figure 133:TWOSAMPLE T TEST COMPARING UNTREATED GROUP AND TREATED GROUP LUTEAL PHASE FREE TESTOSTERONE

Twosample T for FTu vs FTt

```
      N      Mean      StDev   SE Mean
FTu  14      31.7       12.6       3.4
FTt  31      29.7       16.1       3.2

95% C.I. for mu FTu - mu FTt: ( -7.4,  11.4)
T-Test mu FTu = mu FTt (vs not =): T= 0.43  P=0.67  DF=  32
Ftu  Untreated Groups FT
FTt  Treated Groups FT
```

WOMEN

Figure 134:KRUSKAL-WALLIS OF UNTREATED GROUPS PROLIFERATIVE,MIDCYCLE AND LUTEAL PHASE DEHYDROEPIANDOSTERONE SULPHATE



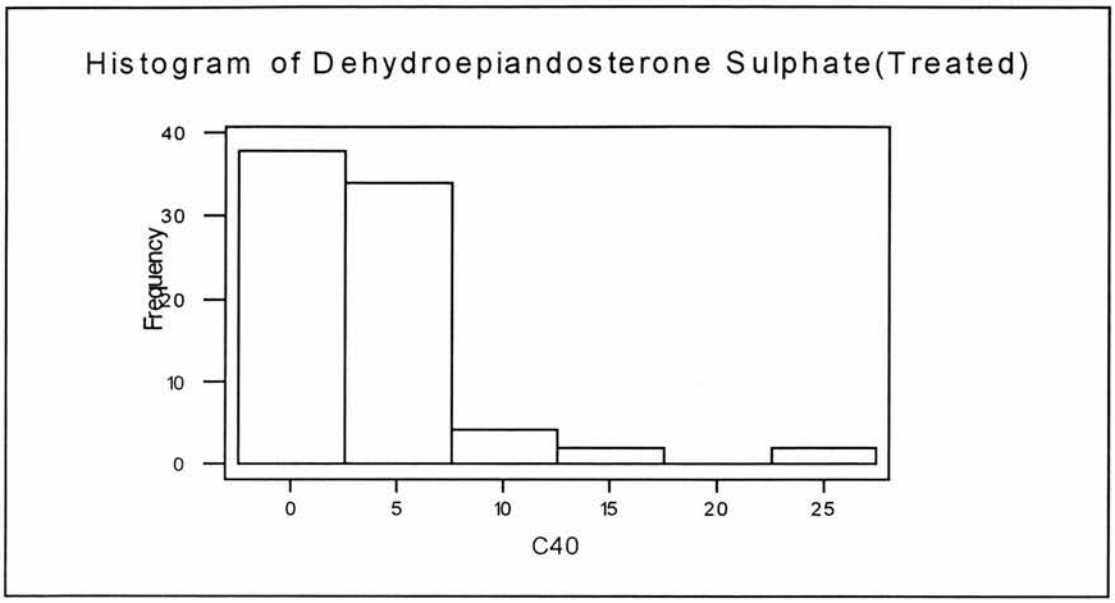
LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
Prolif.	18	3.800	19.6	-1.07
Luteal	14	6.650	24.7	0.97
Midcycle	11	5.300	22.5	0.17
OVERALL	43		22.0	

H = 1.32 d.f. = 2 p = 0.516
H = 1.33 d.f. = 2 p = 0.515 (adjusted for ties)

As can be seen from histogram DHAS values are not normally distributed thus non-parametric test employed.

WOMEN

Figure 135:KRUSKAL-WALLIS OF TREATED GROUPS PROLIFERATIVE,MIDCYCLE AND LUTEAL PHASE DEHYDROEPIANDOSTERONE SULPHATE



88 cases were used

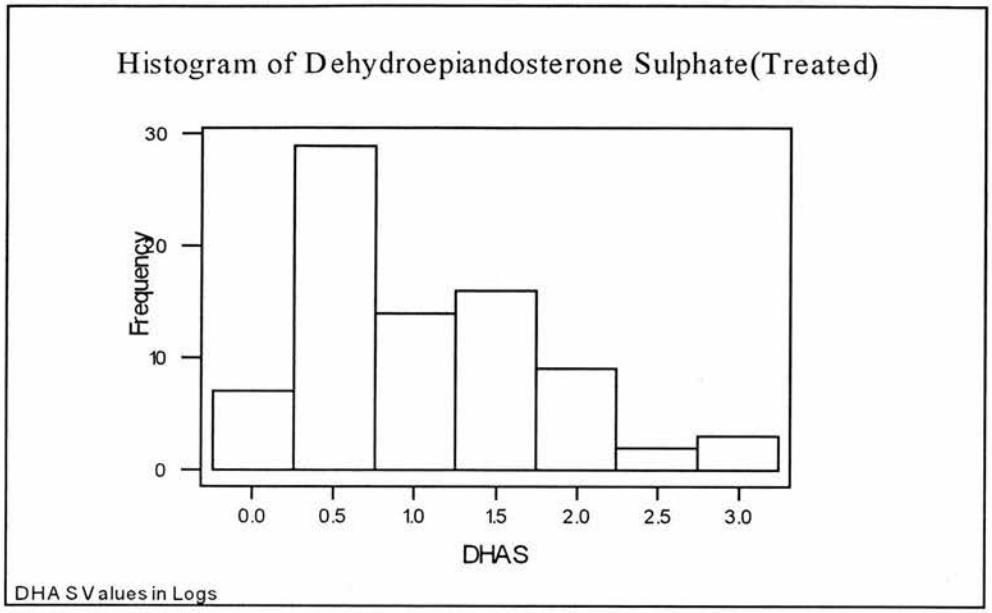
LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
prolif	27	2.000	38.4	-0.51
luteal	31	2.800	41.5	0.27
Midcycle	30	2.600	41.2	0.21
OVERALL	88		40.5	

H = 0.26 d.f. = 2 p = 0.877
H = 0.27 d.f. = 2 p = 0.872 (adjusted for ties)

As can be seen from histogram DHAS values are not normally distributed thus non-parametric test employed.

WOMEN

Figure 136:ANOVA OF DHEAS AFTER LOGARITHMIC TRANSFORMATION




```
Analysis of Variance on DHAS(logs)
Source      DF        SS         MS         F        p
C60          2     0.277      0.138      0.28    0.756
Error       86    37.950      0.493
Total       87    38.227
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level       N      Mean     StDev  ----+---------+---------+---------+--
 prolif     27    1.0296    0.5239   (-------------*--------------)
 luteal     30    1.1391    0.7951          (-------------*------------)
 midcycle   31    1.1713    0.7305             (------------*-----------)
                                     ----+---------+---------+---------+--
Pooled StDev =    0.7020              0.80      1.00      1.20      1.40
```

Fisher's pairwise comparisons

```
   Family error rate = 0.121
Individual error rate = 0.0500
```

Critical value = 1.991

```
Intervals for (column level mean) - (row level mean)
              1          2

     2   -0.5060
          0.2872

     3   -0.5291    -0.4030
          0.2457     0.3385
```

Figure 137:MANN - WHITNEY FOR TREATED versus UNTREATED PROLIFERATIVE PHASE DEHYROEPIANDOSTERONE SULPHATE

```
Untreated          N =  18     Median =       3.800
Treated            N =  27     Median =       2.000
Point estimate for ETA1-ETA2 is       1.200
95.3 Percent C.I. for ETA1-ETA2 is (-0.002,3.499)

W = 456.5
Test of ETA1 = ETA2  vs.  ETA1 ~= ETA2 is significant at 0.0405
The test is significant at 0.0365 (adjusted for ties)
```

Figure 138:MANN - WHITNEY FOR TREATED versus UNTREATED LUTEAL PHASE DEHYROEPIANDOSTERONE SULPHATE

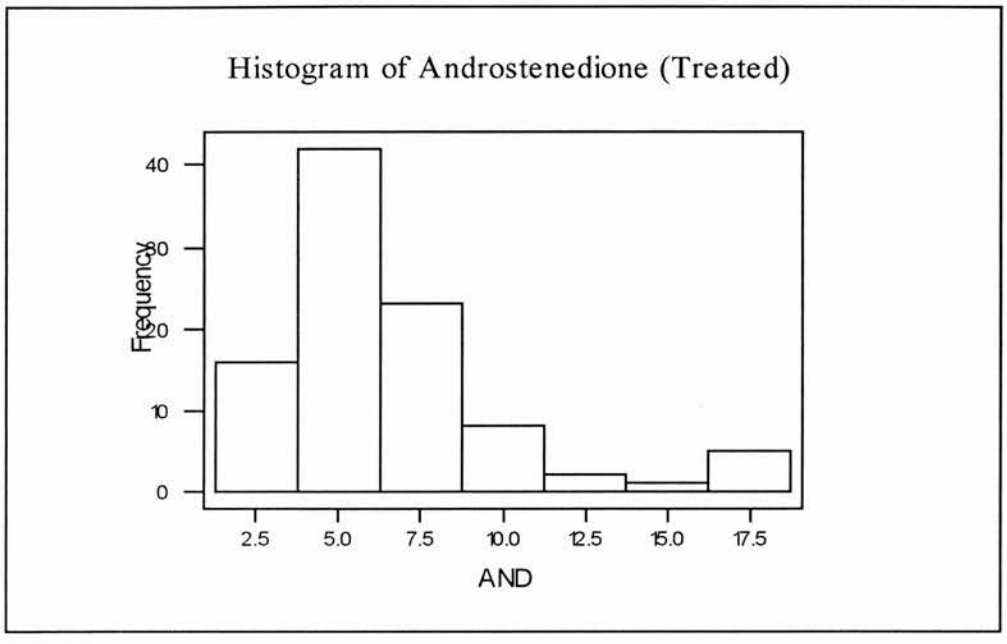
```
Untreated          N =  14     Median =       6.650
Treated            N =  31     Median =       2.800
Point estimate for ETA1-ETA2 is       2.550

95.1  Percent C.I. for ETA1-ETA2 is (0.498,4.900)

W = 382.5
Test of ETA1 = ETA2  vs.  ETA1 ~= ETA2 is significant at 0.0155
The test is significant at 0.0151 (adjusted for ties)
```

Figure 139:MANN - WHITNEY FOR TREATED versus UNTREATED MIDCYCLE PHASE DEHYROEPIANDOSTERONE SULPHATE

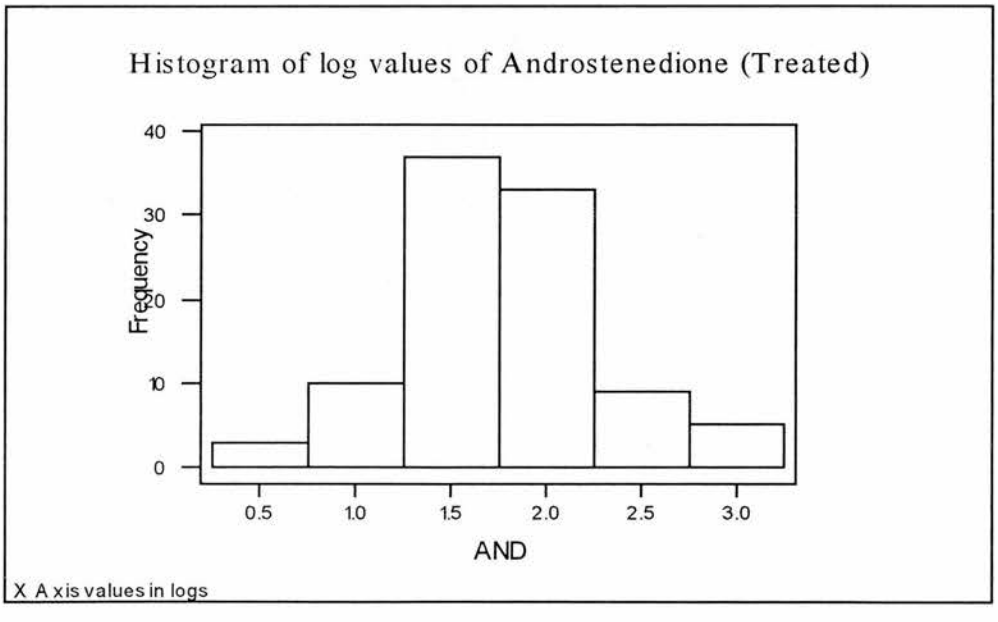
```
Untreated          N =  11     Median =       5.300
Treated            N =  30     Median =       2.600
Point estimate for ETA1-ETA2 is       2.000

95.3  Percent C.I. for ETA1-ETA2 is (0.098,4.402)

W = 306.5
Test of ETA1 = ETA2  vs.  ETA1 ~= ETA2 is significant at 0.0273
The test is significant at 0.0254 (adjusted for ties)
```

WOMEN

Figure 140:ANALYSIS OF VARIANCE OF TREATED ANDROSTENEDIONE



Because values not normally distributed they were transformed to logs as shown below.



WOMEN

Figure 141:ANALYSIS OF VARIANCE OF TREATED ANDROSTENEDIONE

Values in logs

```
Analysis of Variance on Androstenedione(logs)
Source     DF        SS        MS        F        p
C16         2     0.432     0.216     0.92    0.404
Error      86    18.147     0.236
Total      87    18.579
   Individual 95% CIs For Mean
   Based on Pooled StDev
 Level      N      Mean     StDev  ------+---------+---------+---------+
 prolif    27    1.7792    0.4277   (-------------*------------)
 midcycle  30    1.7605    0.5367   (------------*------------)
 luteal    31    1.9201    0.4782               (------------*------------)
                                   ------+---------+---------+---------+
Pooled StDev =   0.4855              1.65      1.80      1.95      2.10
```

Fisher's pairwise comparisons

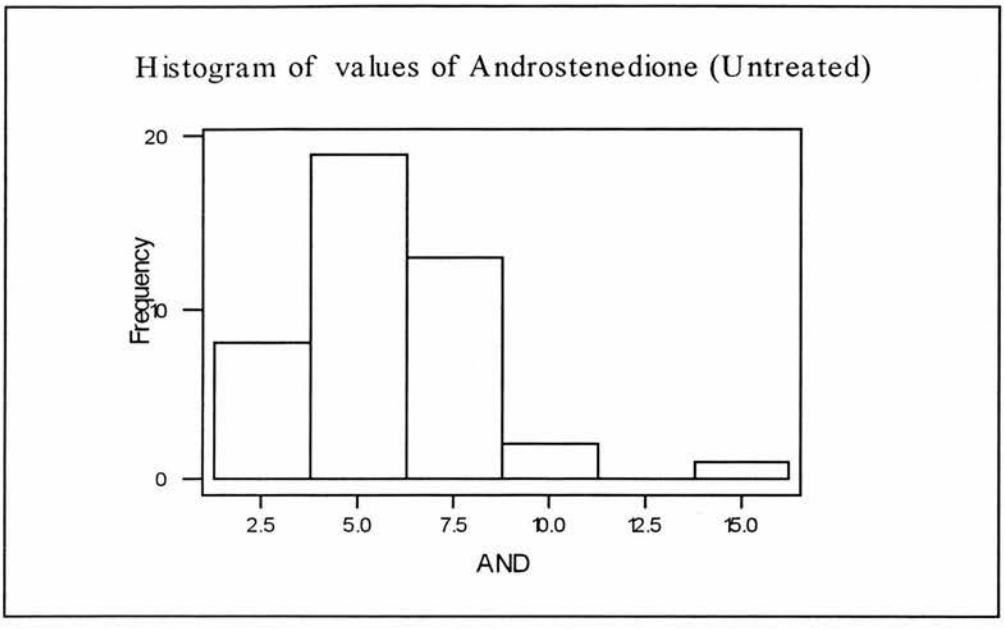
```
   Family error rate = 0.121
Individual error rate = 0.0500
```

Critical value = 1.991

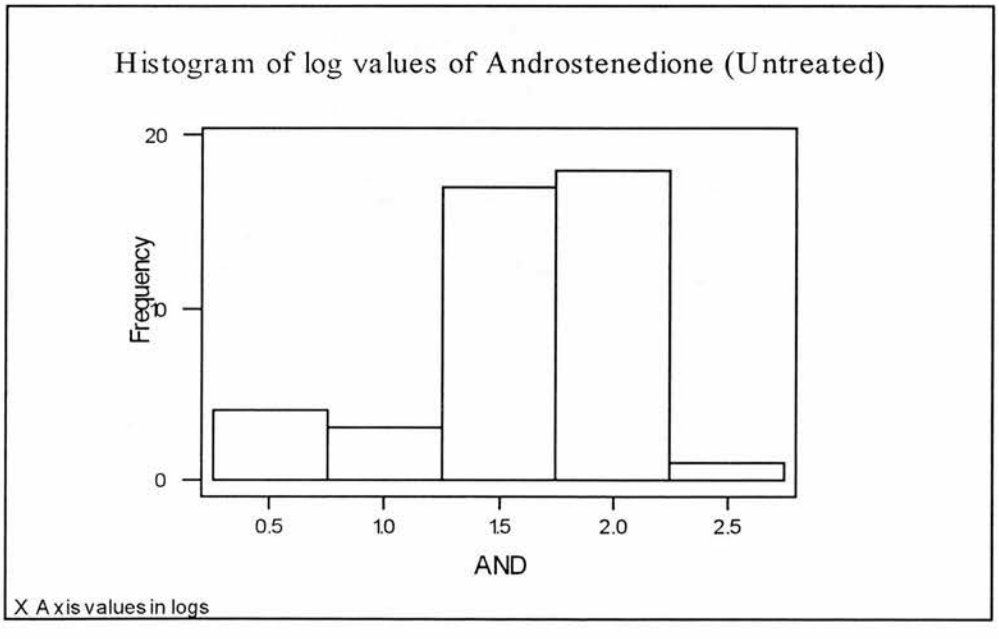
```
Intervals for (column level mean) - (row level mean)
             1         2

     2   -0.2555
          0.2930

     3   -0.4088   -0.4160
          0.1270    0.0968
```

WOMEN

Figure 142:ANALYSIS OF VARIANCE OF UNTREATED ANDROSTENEDIONE



Because values not normally distributed they were transformed to logs as shown below.



WOMEN

Figure 143:ANALYSIS OF VARIANCE OF UNTREATED ANDROSTENEDIONE

Values in logs

Analysis of Variance on Androstenedione(logs)

Source	DF	SS	MS	F	p
C16	2	1.563	0.782	3.91	0.028
Error	40	7.995	0.200		
Total	42	9.559			

Individual 95% CIs For Mean
Based on Pooled StDev

Level	N	Mean	StDev	-+---------+---------+---------+-----
Prolif	18	1.3950	0.4838	(-------*------)
luteal	14	1.6874	0.3305	(-------*-------)
midcycle	11	1.8527	0.5096	(--------*--------)
				-+---------+---------+---------+-----
Pooled StDev =		0.4471		1.20 1.50 1.80 2.10

Fisher's pairwise comparisons

Family error rate = 0.120

Individual error rate = 0.0500

Critical value = 2.021

Intervals for (column level mean) - (row level mean)

	1	2
2	-0.6143 0.0296	
3	-0.8035 0.1119	-0.5294 0.1987

Kruskal-Wallis (Not logs)

LEVEL	NOBS	MEDIAN	AVE. RANK	Z VALUE
1	18	4.200	16.3	-2.51
2	14	5.700	23.6	0.57
3	11	7.000	29.3	2.23
OVERALL	43		22.0	

H = 7.58 d.f. = 2 p = 0.023
H = 7.58 d.f. = 2 p = 0.023 (adjusted for ties)

Figure 144:MANN - WHITNEY FOR TREATED versus UNTREATED PROLIFERATIVE PHASE ANDDROSTENEDIONE

```
UnTreated          N =  18     Median =       4.200
Treated            N =  27     Median =       6.000
Point estimate for ETA1-ETA2 is      -1.750

95.3   Percent C.I. for ETA1-ETA2 is (-3.301,-0.399)

W = 290.0

Test of ETA1 = ETA2  vs.  ETA1 ~= ETA2 is significant at 0.0215
```

Figure 145:MANN - WHITNEY FOR TREATED versus UNTREATED LUTEAL PHASE ANDROSTENEDIONE

```
Untreated          N =  14     Median =       5.700
Treated            N =  31     Median =       5.600
Point estimate for ETA1-ETA2 is      -0.250

95.1   Percent C.I. for ETA1-ETA2 is (-2.299,1.300)

W = 281.0
Test of ETA1 = ETA2  vs.  ETA1 ~= ETA2 is significant at 0.7311 The test is
significant at 0.7310 (adjusted for ties)

Cannot reject at alpha = 0.05
```

Figure 146:MANN - WHITNEY FOR TREATED versus UNTREATED MIDCYCLE PHASE ANDROSTENEDIONE

```
Untreated    N =  11     Median =       7.20
Treated      N =  30     Median =       6.15
Point estimate for ETA1-ETA2 is       0.55

95.3   Percent C.I. for ETA1-ETA2 is (-1.89,2.71)

W = 249.5
Test of ETA1 = ETA2  vs.  ETA1 ~= ETA2 is significant at 0.5964 The test is
significant at 0.5962 (adjusted for ties)
Cannot reject at alpha = 0.05
```