# Sexual Desire and the Deconstruction and Reconstruction of the Human Female Sexual Response Model of Masters and Johnson

# Introduction

Criticism provides one key to science because dissatisfaction with present knowledge and conceptualizations is one mechanism for advancing thought. (Coate, Petersen, & Perry, 1982)

The story of the attainment of accurate human sexual knowledge is long and complex involving not only the historical development of human anatomical studies and of the biology of reproduction (Laqueur, 1990) but also of religion and culture. Bullough (1994), and Porter and Hall (1995) have described in some detail the fascinating account of much of the history while Taylor (1996) has attempted a remarkable survey of its prehistory. The prehistoric portrayals of human sexual organs, especially the female pudenda, are some of mankind's earliest 'artwork' engraved or drawn on the walls of caves or carved artefacts and female figurines (Mcdermott, 1966; Bahn, 1986; Taylor, 1996) although the interpretation of a number of the isolated, triangular images as human pudenda has been criticised as simplistic (Bahn, 1986). The earliest portrayals of human male sexual arousal are also found on the walls of caves or on sexual artefacts. Images of both animals and humans with erect penises have been discovered and in some cases penetration of the phallus into either human female or animal bodies are known. The exact function of the sexual artworks can only be guessed at because we cannot excavate ideas or behaviour but, be they for ritual or magic purposes, they are the earliest graphic characterisations of humankind's need to express their sexual desire.

# The Social Construct Concept of Sexuality

With the invention of writing in Mesopotamia (now Iraq) around 3100 B.C.E., records of what people were doing, their gods and their sexual conduct start to become available. The sexual behaviour described is not always compatible with our modern, politically correct, sensitive ideals but it does show how the individual's most intimate biological acts can be converted by society into specific, controlled behaviours with a social purpose. This feature is the core belief of those that argue that human sexuality is basically a social construct (Foucault, 1980). Thus, while the biology does not

change significantly, the use, interpretation and the meaning of its expression can alter hugely in the context of historical time, social class, ethnic group and religion. Three historical examples illustrate such a concept. The first is from ancient Egypt, where, to prevent outsiders entering the royal family and maintain its claim to rule, pharaohs often married their half-sisters and even their sisters and mothers. Such incestuous marriages were also undertaken by the general population for many discovered letters between husbands and wives begin with 'my dear brother' or 'dearest sister'. The reason for the popularity of these marriages is not yet known but a likely proposal is that they were used to maintain the family's social standing among the varied races and tribes inhabiting the Nile Valley (Miles & Norwich, 1997). The second example, the use of coitus as a sacrament, was described disapprovingly by Herodotus, the Greek explorer and historian of the 5th century BCE, at the Babylonian temple of Ishtar, the goddess of love, in Sumer thus:-'Every woman of the city has to go once in her life to the temple of Innana and give herself to a strange man.....They all sit in rows, with gangways in between so the men can walk through and make their choice. Once a woman has taken her seat she is not allowed to go home until a man has thrown a silver coin into her lap and taken her outside to lie with her....Tall handsome women soon manage to get home again, but the ugly ones stay a long time before they can fulfil the law, some even as long as four years' (!) (Miles & Norwich, 1997). This is a classic illustration of 'sexual scripting', a concept introduced into the field of human sexuality by Gagnon and Simon (1973). Scripts organise and determine the circumstances under which sexual activity occurs. The third and final example comes from ancient Greece where it is clear that some Greek men did not approve of female sexual desire in their wives. Married women were criticised if they showed too much appreciation of the sexual act. According to Xenophon, sexual enjoyment was not the object of marriage, wives were acquired in order to raise a family and not to satisfy lust. The streets and brothels were the places to quench this (Blundell, 1995, p. 102)!

While the ancient civilisations of the Tigris-Euphrates valley, the Indus valley and China had much to say about human sexual behaviour, the knowledge collected by the ancient Greeks, often from Aristotle, Hippocrates, and Galen, to a large extent became for many centuries, the dominant core of Western ideas about human sexuality. Remarkably, many of these ideas, a few accurate but most false, remained until the late nineteenth century when scientific biological inquiry into sex really began to flourish and replace the erroneous.

#### **Orgasm, Coitus and Ovulation**

As only the external body signs of sexual passion were available for observation in antiquity the body responses of the female at orgasm were incorporated into schemes to explain the mysteries of reproduction. An archaic but long running theme, initially created from observing women's whole body movement behaviour during coitus with orgasm, was that the shivering and muscular movements that occurred in women at orgasm was the sign that ovulation had occurred and that the egg was fertilised. In the description of the early authorities, 'semiation' had taken place. Despite the fact that it was shown by many that women could become pregnant without such signs, the reports and arguments were ignored. The preferred wisdom was that orgasmic coitus activated human female ovulation and closed off the womb to stop air entering, for air prevented conception (Laqueur, 1990). It needed many hundreds of years before crucial experiments with dogs showed that ovulation was spontaneous and not related to coitus, moreover it could occur independently of fecundation (von Bischoff, 1843). The final proof however, was much later when the understanding of the phases of the menstrual cycle allowed the collection of actual unfertilised ova from the human oviduct without coitus taking place (Laqueur, 1990). Orgasm was also focused on as the mechanism influencing the sex of the child in Talmudic (4th to 6th centuries B.C.E.) and in Tantric texts (7th to 17th century B.C.E) (Levin, 1987).

While even the brilliant Regnier De Graaf (1668, 1672) believed that ovulation only occurred as a result of coitus, he made two outstanding early contributions into sexual arousal mechanisms. He showed that injecting water into the penis of a human corpse could create an erection indicating its haemodynamic origin and he also described in some detail the lubricatory phenomenon that occurs in the human female vagina during successful psychic and physical sexual arousal. Although a few studies like this had interesting insights, investigating and interpreting the physiological mechanisms by which human males and females become sexually aroused and subsequently attain sexual release is really a remarkably late development. Some highlights of this later history are those of the American obstetrician and gynaecologist Robert Latou Dickinson who conducted research studies between 1890 and 1920 and published them in 1930. His innovative contributions on the sexuality of women and what happened during coitus are summarised in his well-illustrated, classic book on human sex anatomy now some 50 years old (Dickinson, 1949). He was the first to use the insertion of a penis-shaped glass tube into the human vagina to observe what happens during sexual arousal which was initiated clitorally by a vibrator, another first usage. It was a technique that Masters and Johnson developed further by filming through a mechanically thrusting plastic penis inserted into the vaginas of sexually aroused subjects.

The published studies on human female sexual behaviour by Kinsey and his coworkers (Kinsey, Pomeroy, Martin, & Gebhard, 1953) from face-to-face interviews, had two important chapters (14 and 15) that collected and summarised the then known anatomical and physiological facts about the sexual arousal process and coitus. Kinsey and his staff had actually observed volunteer subjects during their sexual arousal in the laboratory for certain aspects of these chapters (Bullough, 1994) but did not publish this fact because of the prevailing hostile scientific and public attitudes to the undertaking of such observations in America. The foundations for the direct observations of Masters and Johnson on human sexual arousal and coitus in the laboratory rested on the bedrock of these previous studies.

#### The Masters and Johnson EPOR Model

William Masters, an obstetrician and gynaecologist, began his studies in 1954 and hired Virginia Johnson (a graduate in music) to help and give him female insights.

After 11 years of observing and studying some 694 human volunteers (382 females and 312 males including 276 married couples) in sexual arousal induced either by masturbation, coitus or by a mechanical artificial penis able to mimic coital movements, they formulated the genital and non-genital changes observed into their concise, arbitrary, four phase EPOR model (Figure 1A) originally described as Excitation, Plateau, Orgasm, and Resolution (Masters & Johnson, 1966). The changes were summarily characterised graphically, using a representation previously popularised by Van de Velde (1926), of a continuous line indicating the intensity of sexual arousal with its climb to a peak and then a subsequent collapse to baseline. Remarkably, in their original diagrams (Masters & Johnson, 1966, Figures 1,1 and 1,2), neither axis of the graph was characterised by a label. While it is clear that the abscissal (x) axis represented 'time', the representation of the ordinate (y) axis is less obvious, it appears to be the 'intensity of the sexual response' but it is unclear whether this sexual response is that of the central excitement (psychological) or of genital excitement (physiological) or a mixture of both (psychological and physiological). Despite these deficiencies the figures were, and still are, reproduced in countless articles and textbooks.

To describe the model briefly, the changes in the Excitement phase developed from any source of somatogenic or psychogenic sexual stimulation. If the continued stimulation was effective, the sexual tensions were intensified and the subject entered the Plateau phase. Continuation of the stimulation moved the subject into

	Excitation (E)	Plateau (P)	Orgasm (O)	Resolution (R)	A Masters & Johnson (1966)
Desire (D)	Excitation (E)		Orgasm (O)	Resolution (R)	B Kaplan (1979)
Desire (1)	W Torona M Markov David	tation re (2)	Orgasm	Resolution	C

Fig. 1. The development of the linear sequence, human sexual response model from the original EPOR model of Masters & Johnson (1966) (A), through the DEOR model of Kaplan (1979) (B), to the proposed modification (C) with Desire phase 1 (before initiation of the Excitation phase) and Desire phase 2 during Excitation phase. See text for details.

the Orgasmic phase the duration of which was relatively short and led to the dissipation of the vasocongestion and myotonia induced previously by the sexual arousal accompanied usually by the intense feelings of ecstasy. This then leads into the Resolution phase where the tension loss is the reverse of the original Plateau and Excitement phases back to the unstimulated state. The model was linear and incremental and could be applied to both heterosexual and homosexual sexual arousal. If, during the Excitement phase, the sexual stimuli was not sufficient to attain the Plateau phase and subsequently the Orgasmic phase, the subject's Resolution phase was slower and the return to the unstimulated state took much longer to attain. The descriptive EPOR model became adopted, initially with very little criticism, by sexologists, clinicians, sexual therapists and more slowly even by reproductive physiologists. Because of its ability to be applied to the male and female, to heterosexual and homosexual subjects and to pregnant and menopausal women it dominated sexology and was used in 1980 as the basis by the American Psychiatric Association's Diagnostic and Statistical Manual for diagnosis of sexual dysfunctions. Even today it is still uncritically used in its initial form in textbooks and articles. Few workers, and surprisingly not least the authors themselves, have taken seriously the original statement by Masters and Johnson (1966) in their introductory chapter that 'It should be emphasised that these patterns are simplifications of those most frequently observed and are only representative of the infinite variety in female sexual response'. Application of new recording and investigatory techniques is now allowing researchers in the field to objectively substantiate certain aspects of this statement (see review by Levin, 1998).

## The Problem of the Desire Phase

Dissatisfaction with aspects of EPOR model, however, was voiced by Hoon (1979) in relation to definitional reliability (there was no interobserver agreement about the changes observed) and sequential reliability (Guttman scaling was not applied) and by others (Gagnon, 1977) that the experimental population chosen by Masters and Johnson to observe in the laboratory were a highly selected orgasmic group who did not mind being watched during their sexual activities and who could arouse themselves at a specific appointed time, presumably without having spontaneous sexual desire, for scientific or altruistic (or even exhibitionistic?) purposes. Tiefer (1991, 1994) has also used similar points in her critique of the EPOR model as a self-fulfilling project using selected participants with a high interest in sexuality and who were 'orientated' or 'desensitised' to the laboratory environment or equipment by training sessions. While all these criticisms are valid, the type of studies undertaken by Masters and Johnson simply cannot be made on a statistical random sample of the population. A much more important criticism came from the American sex therapist Helen Kaplan (1977). She was seeing in her sexual treatment clinic numbers of female subjects who professed to have no desire for sexual relations even with their loved partners. Previously, Goldberg (1973) had observed a number of male patients with the same problem- lack of sexual desire.

Because of her patients, Kaplan (1977) proposed that as they had an absence of desire to undertake sexual activity then there must be a phase in the normal sexual scenario that initiates or activates females to want to experience the excitation phase. She called this new phase the 'desire phase'. Helen Kaplan (1979) defined sexual desire (or libido) as 'specific sensations which move the individual to seek out or become receptive to sexual experiences'. These sensations, she argued, 'are produced by the physical activation of a specific neural system in the brain' but what these neural systems were could not be specifically delineated although suspected to be located in the limbic system.

Human sexual desire is complex and is not a simple entity, it can change during the sexual scenario experienced by a person. Four, practically serial subcategories of sexual desire usually occur during a heterosexual encounter, i) wanting sexual activity/arousal to occur in one's body, ii) wanting to create sexual arousal in another's body, iii) wanting coitus to occur, and iv) wanting orgasm to occur. It is possible to have sexual desire without sexual arousal and sexual arousal without sexual desire. The pleasure of sexual arousal is not always welcome. In some circumstances sexual arousal can lead to dismay and even to disgust especially if thought inappropriate. Some authors have argued that it is difficult to distinguish between sexual desire and sexual excitement (Everitt & Bancroft, 1992). They stated that 'to make an operational distinction between sexual desire and sexual excitement quickly comes to grief. When does desire become excitement? Is a sexual thought or fantasy a stimulus or a response, or both?' Levin (1994) has discussed a number of these problems in a review on human male sexuality and created a simple sexual scenario where it was possible to distinguish between sexual interest, desire and arousal, albeit fuzzily. Stoller (1976), an American psychoanalyst, ruminated at some length on the extensive lexicon used to describe sexual excitement and the semantic difficulties involved if it was equated to sexual desire. He argued that this can apply to a state in which the body's anatomy and physiology are not yet engaged (presumably he means here 'overtly').

While the discrete clinical entity of insufficient sexual desire had first been recognised by sex therapists in the 1970's it was not until the third edition of the DSM (DSM-111; American Psychiatric Association, 1980) that desire problems were included as the independent entity 'Inhibited Sexual Desire Disorder' defined as 'persistent and pervasive inhibition of sexual desire'. A later elaboration (DSM-111R (American Psychiatric Association, 1987) divided the disorder into i) Hypoactive Sexual Desire Disorder and ii) Sexual Aversion Disorder. By 1988 it was possible for Leiblum and Rosen to edit a book (Sexual Desire Disorders) in which some nineteen authors discussed a range of treatments for disorders of human sexual desire. It is interesting to note that Masters and Johnson had not modified their human sexual response model to include the desire phase as late as 1985 (Masters, Johnson, & Kolodny, 1985). Subsequently, the Masters and Johnson Institute's therapeutic approach to sexual problems agreed with the importance of desire phase difficulties as a legitimate clinical problem and created a treatment model for inhibited sexual desire (Schwartz & Masters, 1988).

## The Problem of the Plateau Phase

The arbitrary division between the Excitement phase and the Plateau phase was clearly highlighted by Robinson (1976). There seems to be very little difference between high levels of sexual excitement and the so-called Plateau Phase, they both exist on a continuum of ever-increasing sexual tension as long as the sexual stimulation is maintained and accepted. There appears to be no discontinuity as occurs with the Orgasmic phase, when both smooth muscle and pulsatile striated contractions are initiated. In the Plateau phase, while vivid colour changes appear to take place in the labia minora (created by changes in blood content and its desaturation), the glans of the clitoris moves back under its hood, the vasocongestion of the outer third of the vagina becomes maximal and causes partial occlusion of the lumen (creation of the orgasmic platform), and the upper vaginal tenting and expansion is maximised. None of these changes are really anything more than further extensions of the Excitement phase changes and can be easily classified as late excitement phase changes. There appears little need to create another phase. Moreover, the terminology of the Plateau phase implies that the sexual tension is stable and level (viz plateau) whereas the exact opposite is true, it is actually building up to initiate the final explosive orgasmic release. Robinson (1976) suggested that the naming of the plateau phase was to coincide with the creation of the outer vaginal orgasmic platform ( = plateau) but there is no real evidence for this speculation. It should be remembered that during the formulation of the EPOR model, Masters and Johnson had no objective, quantitative measure to use as an indicator of female genital sexual tension apart from visual observations of the colour of the labia. All the techniques for the quantitative estimates of vaginal blood flow (a good index of the arousal state of the female) and oxygen tension, were developed in the 1970's well after the publication of their four phase model (see Levin, 1980, 1981, 1983, 1991, 1994, 1997; Rosen & Beck, 1988).

## **DEOR- the Modified Human Sexual Response Model**

In Kaplan's (1979) modified human sexual arousal model there was first a desire phase (D-phase) then the excitation phase (E-phase). Because of the lack of support for distinguishing the Plateau phase from the excitation phase, as discussed previously, the next phase becomes the Orgasmic phase and then the Resolution phase. We thus now have a DEOR model replacing the EPOR model (Figure 1B). The diffusion of this new model into the general literature and textbooks was slow but gradually the concept of the Desire Phase gained acceptance and it became practically de rigeur to include it as an important aspect of human sexual arousal. It added a psychological component to what was a mainly peripheral, organ-response dominated model. As related previously, its origin arises from studying female patients who lacked sexual desire (i.e., those who were sexually dysfunctional). Today, it is known that studies using the female clinic population do not necessarily represent the 'normal' female population. Remarkably, there did not appear to be any study in 'normal' or sexually functional control subjects that such a phase existed before the excitation phase. There is sparse literature about the topic, neither Bullough (1994) nor Porter and Hall (1995) have an entry for human female sexual desire in their indices or mention studies on it in their histories. The idea that the human female had any sexual desire is again a relatively late concept. While on a simple evolutionary basis it would appear obvious that the reason for female sexual desire is to make her attempt mating to reproduce the species, Laqueur (1990) ably summarised the early literature that questioned the 'very existence of female sexual desire'. Reports and writings from many physicians were based on nothing more than their own opinions of the William Acton-type that 'the majority of women are not much troubled by sexual feeling of any kind' (Acton, 1865, pp. 112). In France, at the fin-de-siecle, 'female sexual desire was considered to be virtually non-existent in the virgin, but to be a force that could be awoken by male desire and must then be carefully controlled in the interests of male well-being and the institution of marriage' (Holmes, 1998). No scientific epidemiological information about the incidence of female sexual desire was available in the nineteenth century.

What information then is there to support the assumption that in normal sexually functional females there is a so-called desire phase preceding the excitation phase?

## **Desire Phase Studies**

#### Sexual Desire During the Menstrual Cycle

A number of studies have been conducted on the sexual feelings and sexual activity of samples of heterosexual females (usually with partners) over the menstrual cycle especially to see if there is a peak of 'spontaneous' sexual desire (usually for coitus) at or before ovulation and to try and correlate these with their different hormone levels. The hypothesis being tested is that it would be expected that such females would desire sex leading to possible coitus at this time to enhance the chances of being fertilised, i.e., maximise reproductive success. Technical difficulties in standardising and comparing menstrual cycles of different lengths, of identifying the precise timing of ovulation in the cycle, of measuring total plasma hormone levels (bound and unbound), and of choosing which measure(s) of female sexual activity to monitor have created a confused literature (Meuwissen & Over, 1992). Interestingly, many studies showed that there was a clear increase in sexual desire perimenstrually. The usual arguments are that this arises because, i) males least liked to partake of sexual activity with menstrous females and this temporary lack of male-activated sexual activity with the female stimulated desire, and ii) the low level of androgens in the female plasma perimenstrually was least opposed by oestrogen and progesterone, both normally antiandrogenic. While androgens are often quoted as playing a hormonal influencing role in female sexual activity (Bancroft, 1984; McCoy, 1992) the scientific evidence leaves much to be desired.

One of the better controlled and thought-out studies was that of Adams, Gold, and Burt (1978) who monitored female sexual activity that was initiated by the female *per se* rather than by her male partner. When this was done an obvious combined

peak of autosexual and heterosexual behaviour was noted but it was the former that was creating the significant increase. Some have interpreted this study as showing that enhanced 'female sexual desire' was observed around ovulation and it was absent in anovulatory women that were taking the steroid contraceptive pill. While a reanalysis of the data by Kolodny and Bauman (1979) could not confirm the enhancement at ovulation a study by Harvey (1987) also found that female initiated sexual behaviour peaked at the ovulatory phase.

Wood (1994) reviewed the various studies in relation to changes in coital frequency around midcycle and proffered that there were 'tantalising hints' of midcycle increases but that even if these changes were real the available samples may all be too small to reject the null hypothesis of uniform coitus over the inter-menstruum.

These studies of sexual-desire changes during the menstrual cycle have always used highly selected groups of women (and their partners) who agreed to enter a recording programme detailing their sexual activities and even having to have blood taken. They are clearly not a representative sample of the general female population. If one is ultracritical, the validity of the data obtained from them could be classed as suspect!

#### Desire Assessed by Questionnaire

Another way of studying the 'spontaneous' desire phase is to obtain a statistical representative sample of the general female population who are sexually active and orgasmic and ask whether they spontaneously experience sexual desire. Garde and Lunde (1980) undertook such a careful and extensive sexological study in 1976 from a random sample (n=225) of Danish women all born in 1936. The face-to-face interviews were structured and conducted by the female physician. Many aspects of sexuality (170 questions) were asked. Practically all the women (96%) had at some time experienced at least one orgasm, approximately 19% always attained orgasm during coitus, 61% almost always or often and 15% less frequently. Only 0.9% reported that they never had orgasm during coitus but did from masturbation and cunnilingus. In regard to their sexual desire (in this study designated as libido) they were asked to characterise it under two domains, Degree 1: the woman's own, spontaneous desire for sexual activity irrespective of which form and irrespective of whether the desire is satisfied, Degree 2: the woman's own desire arising from some form of stimulation, e.g., by a partner. They found that approximately 68% of the women 'knew the experience of spontaneously feeling libido (degree 1) while 12% had never experienced any libido. These figures surprisingly indicate that some 32% of 40 year-old sexually-experienced and functional Danish women never experienced spontaneous sexual desire. In a more recent sex survey held in America Michael, Gagnon, Laumann, and Kolata (1994), using a random sample selection, reported that in response to the question 'During the last twelve months has there ever been a period of several months or more when you lacked interest in sex?' one out of three women agreed (compared to one out of six men) which was interpreted as some 33% of women being uninterested in sex, a figure agreeing remarkably with that found in the described Danish survey. Thus the admittedly

extremely limited data suggest that approximately 33% of sexually functional women do not appear to experience a spontaneous desire phase. Should this make us question the centrality of a spontaneous desire phase to the human female sexual response model?

Another difficult feature about the desire phase is its positioning in the sequential models of sexual response. Kaplan (1979), and subsequently others, placed it before the excitation phase but an important question arises from this decision namely, does the desire phase always exist before the excitation phase?

## The Position of the Desire Phase in the Sequential Female Sexual Arousal Model

Perhaps the difficulty in answering the question is in thinking that there is only one type of activation of female desire. The spontaneous phase (desire 1), created endogenously, clearly has to be placed before the excitation phase (Figure 1B) but a second desire phase created by exogenous stimuli (desire 2) could well be positioned during the excitation phase (Figure 1C). This is not meant to imply that there are two separate desire mechanisms acting independently in the brain but rather that there is one mechanism that can be activated at different times of the to-be-experienced and experienced sexual activity.

Placing the spontaneous or endogenous desire phase (desire 1) before the excitation phase has an obvious utility. The woman initially feels a spontaneous desire to be sexually active and then undertakes either self-stimulation or partakes of sexual activity with a partner. The obvious question that arises is what causes the initiation of the spontaneous or endogenous sexual desire? The emphasis here is on the words 'spontaneous' or 'endogenous'. It is possible to postulate a galaxy of stimuli that could create female sexual desire. Previously experienced odours, tastes, images, words, music, thoughts, fantasies- all can initiate centrally the activation of spontaneous sexual desire. In certain respects if these stimuli come from being activated by outside stimuli, then it would be inaccurate to call the activated desire spontaneous or endogenous but the distinction to be made here is perhaps one of psychological activation rather than physical (Figure 1C). Herz and Cahill (1997) explored the use of sensory information in sexual behaviour as a function of gender. When not engaged in sexual activity, visual experience, both imagined and real, for both males and females was the most sexual arousing. The next most arousing for males were hearing human sexual sounds and tactile stimuli. For females, after visual stimuli, the next most arousing stimuli were touch and music, hearing human sexual sounds was the least arousing sexual experience. It is interesting in this context to realise that the vast majority of aural, sexually explicit sound tapes of human sexual activity are made for, and bought by, men (Levin, 1992).

### Is there a Brain Centre for Sexual Desire?

Remarkably little is known about the underlying neurobiological basis of human sexual desire, arousal and orgasm especially at the brain level. The earlier studies have been reviewed by Levin (1992, 1994). Brain imaging in specific human sexual states is only just beginning to be undertaken (see the conclusion for references).

The two simplest neuronal 'desire models' that can be described are i) of a set or pool of neurones that slowly become activated and when enough are so primed the summated output they can create finally activates a feeling of want or sexual desire or ii) a pool or set of neurones that activate desire when allowed to fire off but are held in check by inhibitory inputs. In the former model, stimulatory inputs are fed to the desire locus while in the latter, stimulatory inputs are fed to the inhibitory locus which when inhibited itself then allows the desire centre to act. At present there is no experimental observations to accept either model or their combination. While the latter one appears more complicated (inhibition of an inhibition), it should be remembered that in the sexual mechanism of penile erection the engorgement of the flaccid organ comes about partially by the inhibition of the inhibitory sympathetic activity (outflow) that constricts the organ's blood vessels. It is also known that damage to certain areas of the brain (temporal lobes, frontal lobes, septal nuclei, and limbic system) can lead to hypersexual behaviour as if these areas held in check the area for sexual desire and if they become damaged release this function from regulating control (Malatesta & Robinson, 1995). Under normal circumstances do sexual stimuli have to inhibit these inhibiting areas to activate sexual desire?

Some authors propose that a physiological substrate in the brain for desire may be non-existent (Rowland, 1995) arguing that desire may simply entail a state of high sensitivity (low threshold) in the pathways involved in arousal. The concept of a single desire site in this argument is too simplistic as the interaction of a number of brain areas is essential for sexual response such as sensory, information processing, motivational and consummatory aspects all have to be integrated to give the final sexual behaviour. In this context another feature of the brain may also be involved. The inability of the human mind to recreate exactly the pleasure felt by sexual arousal, especially the ecstasy of orgasm, although it can be remembered, must be part of the equation of sexual desire in many people. Once the waters of sexual pleasure have been tasted the urge to repeat the experience (drink at the same fountain!) is likely to occur.

#### **Hormones and Desire**

Hormonal levels, especially androgens (Salmon & Geist, 1943: Segraves 1988a), could interact centrally in the brain to activate desire, or to increase the intensity of the gratification or pleasure and/or with the genitals by possibly changing the sensitivity of their touch and sensory receptors and /or the way that the afferent signals are processed centrally. Androgens can also change the neuroamine levels in the brain, transmitters well-known to affect sexual behaviour in animals but less well studied in humans (Fabre-Nys, 1998; Melis & Argiolas, 1995). The nature of the genito-pelvic afferent nervous traffic can also be affected by hormones changing genital blood flow and creating vasocongestion.

### Erotic Sites for Creation of Spontaneous Sexual Desire

## Genitopelvic Afferent Input to the Brain

How important is the genitopelvic afferent input to the brain in relation to the creation of spontaneous sexual desire? The first study was by Money (1960) who interviewed paraplegic men (n=14) and women (n=3) with clinically verified spinal lesions so that no genito-pelvic afferent signals reached the brain. He concluded that 'Quite unequivocally, sexual desire was not experienced by these paraplegic patients as it was before the injury and they had no genitopelvic gratification'. He postulated that 'In the ordinary course of events, in uninjured people, it would appear that the generation of messages locally in the genitopelvic part of the nervous system is of basic significance in the generation of the subjective, waking experience of sexual urge and gratification'. This suggests that constant genitopelvic afferent inputs to the brain are essential to maintain the mechanism(s) for spontaneous sexual desire. Later Money (1977) reinforced the importance of genitopelvic afferent input for maintaining normal sexual desire. He pointed out that it was not the loss of genital tissue that counted, for men who had amputated penises because of cancer or accident trauma still had sexual desire and maintained orgasm while in sex reassignment operations for male transsexualism (men into women) the corpora are extirpated and only the skin is retained as the lining of the new vagina (Jones, Schirmer, & Hoopes, 1968). After operation such transsexuals living as females still report experiencing orgasm. Women who have ablation of genital tissue (resection of the vulva or from extirpation of the clitoris) for cancer can still retain erotic sensation and orgasm. Finally, in 1993, Money revisited the sexuality of paraplegic subjects. He had reported in his 1960 study that they could experience orgasms in their dreaming sleep and named them 'phantom orgasms'. Its occurrence was infrequent and it progressively disappeared within a maximum of 2 years after the spinal cord injury. He claimed that the 'long term continuity of the existence of orgasm in the brain is contingent on the continuity of its synchronous existence in the pelvic genitalia'. More recent laboratory studies on the sexuality of spinal cord injured women (Sipski & Alexander, 1995) do not appear to show the same complete depression of sexual desire that Money described in his 1960 subjects. A number of spinal cord injured females appear to enjoy stimulating themselves sexually to experience what they claim is orgasmic responses. They are also described as performing some type of genital stimulation to achieve orgasm regardless of whether their injuries were complete or incomplete. At first this seems a bizarre finding but presumably the sensory feedback from touching their genitals (wetness, hotness, swollenness?) is obviously a significant feature for their arousal. In all of these studies with spinal cord injured women the absolutely crucial feature is the extent of the severity of the spinal injury, even a small amount of undamaged cord could allow neural traffic from the genitals to the brain. Unfortunately, the detailed neurological work up of the women with spinal cord injury is not always forthcoming. More recent studies (Komisaruk, Gerdes, & Whipple, 1997) have used the definition of 'complete spinal cord injury' (SCI) as that defined by the American Spinal Injury Association (1992) viz i) no awareness of pinprick and light

	Putative mechanis	ms of activation by		
Primary Erotic pressure / friction sites	Foreplay / Masturbation	Coitus	Key references on structure / function	
clitoris (external) (glans / shaft)	surface friction of glans	<ul> <li>i) labial hood traction</li> <li>over glans (unlikely),</li> <li>ii) friction from penile</li> <li>shaft in high ventral-</li> <li>ventral coitus</li> </ul>	Masters & Johnson (1966) Alzate (1980)	
clitoris (internal) – shaft, crura, around urethra (?)	pressure on anterior vaginal wall	penile thrusting pressure on anterior vaginal wall	O'Connell et al. (1998)	
periurethral glans = triangular area between apex of vaginal introitus and clitoral glans surrounding urethral meatus (corpus spongiosum?)	surface friction by rubbing	penile thrusting moves shaft over area pushing it in and out of vagina creating friction	Levin (1991; 1998) Turnhout et al. (1995)	
G-spot – around base of bladder / urethra = periurethral or paraurethral glands (?)	stimulation of anterior vaginal wall with deep pressure stroking	penile thrusting of anterior vaginal wall with deep pressure stroking	Grafenburg (1950) Hoch (1986) Alzate et al. (1989) Lenck et al. (1992) Levin (1998)	
Halban's fascia – space between trigone and urethra fibro-elastic tissue with rich blood supply and innervation (pseudo-corpuscular nerve endings)	(corpus spongiosum?) stimulation anterior vaginal wall with deep pressure stroking	penile thrusting on anterior vaginal wall with deep pressure stroking	Minh et al. (1979) Hoch (1986)	
urethra- submucosal erectile tissue (corpus spongiosum?)	stimulation anterior wall by deep pressure stroking	penile stimulation anterior wall by thrusting deep pressure stroking	Grafenberg (1950) Levin (1992; 1998)	
Cervix / uterus	jostling of cervix up and down and to and fro to make uterus rub the peritoneum	deep insertion penis jostling cervix to make uterus rub the peritoneum especially in rear entry or sideways rear entry	Singer (1973) Riley et al. (1992)	
labia (especially minora)	surface friction	surface friction from penile thrusting	Masters & Johnson (1960) Hoch (1986)	
vaginal or vestibular bulbs (clitoral bulbs)	pressure	penile friction and pressure	Masters & Johnson (1960) O'Connell et al. (1998)	

The various primary erotic pressure and friction sites present in the human female genitalia and their putative mechanisms of activation by either foreplay/masturbation or in coitus. Many other sites are also capable of creating or enhancing sexual arousal (viz nipples, breasts, perineum, skin areas such as underarm, back of knees, inside of thighs, perianal) but these have not been included.

touch below the level of the SCI, ii) no awareness of digital rectal stimulation, iii) and no voluntary movement below the level of the SCI.

The hyperaesthesia of the body that often occurs above the lesion of the spinal cord (Higgins, 1979) is exemplified in the single case report by Kolodny, Masters, Johnson, and Biggs (1979) about an able-bodied woman who participated in their laboratory studies on human sexual responses and later sustained a spinal cord injury (complete lower motor neuron lesion at T-12) that made her lose all pelvic sensation. As able-bodied she derived little erotic pleasure from breast stimulation. After the injury however, her breasts became increasingly sensitive to erotic stimulation and after 6 months she became orgasmic on their stimulation. Such stimulation in the laboratory revealed that there was no significant degree of pelvic vasocongestion but her lips were congested! The transference of erotic sensitivity from genitals to breasts is similar to that reported by African women who had been circumcised (see Megafu (1983) in section below on clitoris).

In the female, the numerous genito-pelvic and erotogenic sites that can feed afferent stimuli to the brain are shown in Table 1. Of these, probably the most important organ for activating arousal normally is the clitoris.

#### The Clitoris

The knowledge that women could experience sexual pleasure was a commonplace in antiquity. The clitoris was claimed to be first described accurately by Readolus Columbus in 1559, the successor to the chair of anatomy at Padua previously occupied by Vesalius. He described it as 'pre-eminently the seat of woman's delight'. Like a penis 'if you touch it, you will find it rendered a little harder and oblong to such a degree that it shows itself as a sort of male member'. Kasper Bartholin (1611), however, the Danish anatomist, later criticised this appropriation of the discovery of clitoris since, according to him, it had been known to everyone since the second century! The term first appears in English in 1615 described by the anatomist Helkiah Crooke. Its ability to arouse women if stimulated was known to be important as it 'makes women lustful and take delight in copulation' without which 'they would have no desire, nor delight nor would they ever conceive' (Sharp, 1671). Freud's (1905/1930) concept of the function of the clitoris over two centuries later was that its 'task namely, of transmitting the excitation to the adjacent female sexual parts, just as – to use a simile – pine shavings can be kindled in order to get a log of harder wood on fire' exposed his bias that sexual arousal by the clitoris was facile but by that of the vagina not so.

Clitoral development. Today we know that the human external genitalia are identical until week 8 of gestation. The penis develops from the genital tubercle under the influence of  $5\alpha$ -dihydrotestosterone converted by the local cellular enzyme  $5\alpha$ -reductase from testosterone secreted by the foetal testis. In the absence of the foetal testis and its androgen secretion, the tubercle develops into a clitoris but it can still respond to androgenic stimulation. Contemporary women athletes who have taken large doses of androgenic steroids to build up muscle and strength report anecdotally

that the androgenic stimulation of their clitorises creates a hypersexual condition where they are near orgasm and have much sexual desire. But the doses taken are well above the physiological levels. It is interesting to note that while androgen receptors are present in large quantities in the immature penis they decrease with age in the rat and human (Rajfer, Namkung, & Petra, 1980) and are found in low amounts in the adult human penis (Nonomura, Sakakibara, Demura, Mori, & Koyanagi, 1990). This probably accounts for the relative lack of response of the penis to androgen stimulation after puberty (Shabsigh, 1997) although experiments by Baskin, Sutherland, Disandro, Hayward, Lipschutz, and Cunha (1997) using grafted human foetal penises in athymic nude mice indicated that mechanisms other than loss of androgen receptors were also a factor in causing the inhibition of adult penile growth to circulating androgens. The refractory response of the adult penis to androgen is probably essential to prevent it from growing inconveniently larger and larger with age under the constant androgen stimulation (despite the present day popular advertising campaign concept that 'size matters'!). This is not the case in the female.

*Mature clitoral response to androgens.* Although as stated previously, the clitoris develops from the same tissue anlagen as the penis, it continues to grow and respond to androgens throughout the lifespan of the female (Huffman, 1969). It does not lose its androgen receptors after puberty. The external clitoris of the older woman is larger in size than when she was younger. The increase in size of the clitoris while stimulated by the low levels of androgen secreted by her adrenals is presumably held in check by the high levels of oestrogens, at least while she is premenopausal.

Despite ancient wisdom that 'It is reasonable both to reason and authority that the bigger the clitoris in woman, the more lustful they are' (Culpepper, 1675, p. 22) it is now usually accepted from cross-sectional studies that external clitoral size *per se* is no arbiter of its sensitivity to tactile stimuli, a small clitoris can be as effective in arousing a woman as a large one (Dickinson, 1949; Masters & Johnson, 1966). What is not so secure is whether clitoral sensitivity to tactile stimuli in an individual is enhanced over her lifetime, as it grows bigger and presumably has more area to be stimulated. Baily (1973) claimed an almost linear correlation between clitoral sensitivity and orgasmic capacity but no data were shown. Riley, Riley, and Brown (1986) also reported that women with poorly developed external genitalia, in particular hypoplastic clitorises, show low levels of sexual interest. They only experience sexual arousal and achieve orgasm with difficulty. Testosterone treatment is known to enhance clitoral size and sensitivity, treating these subjects with androgens was reported to be helpful.

#### Clitoral Anatomy

Although the clitoris has been known for hundreds of years the details of its erectile tissue structure is still under investigation. Turnhout, Hage, and Diest (1995) confirmed by dissections in fresh cadavers that the bilateral vestibular bulbs on each side of the vagina terminate into the glans of the clitoris. According to them this tissue is homologous to the corpus spongiosus of the penis. Another study by Toesca, Stolfi,

and Cocchia, (1996) has indicated that the corpora cavernosa of the clitoris is essentially similar to that of the penis except that there is an absence of a subalbugineal layer interposed between the tunica albuginea and the erectile tissue and this tissue has a rich venous plexus. In the penis this tissue becomes engorged with blood during sexual arousal and becomes compressed against the unyielding tunica albuginea creating penile rigidity (= erection). The absence of this plexus in the clitoris suggests that while the organ can become tumescent (engorged or swollen) it cannot, like the penis, become stiffly erect (rigidity).

The more recent cadaveric clitoral dissections by O'Connell, Hutson, Anderson, and Plenter (1998) received unprecedented publicity when a popular British weekly science journal over enthusiastically reviewed the paper and claimed in an editorial that 'doctors have never really understood the gross anatomy of the clitoris let alone the fine details of its blood and nerve supply' because they 'have either been too prudish to examine female anatomy in detail, or just didn't think it mattered' (Editorial, 1998; Williamson & Nowak, 1998). In fact the concept of an extensive internal clitoral erectile tissue was published previously in a series of drawings characterising the female genitalia (Dawner & Chalker, 1980). The O'Connell et al. (1998) study involved the dissection of 10 female corpses ranging in age from 22 (n=2, premenopausal) to 88 years old (n=6, postmenopausal). They reported that while the glans and frenulum of the clitoris is accurately described in textbooks, the internal body of the clitoral erectile tissue was poorly characterised. They claimed that the clitoral structure was a triplanar complex with the midline corpora (1-2 cm wide and 2-4 cm long, lying in the median sagittal plane) giving rise to the paired crura (5-9 cm long, lying parallel to the ischiopubic rami) and separate bulbs (3-7 cm long, crescentic or triangular, sitting posterior to the corpora) the urethra lying surrounded by this complex of erectile tissue. The so-called vestibular bulbs do not form the core of the labia minora but are part of the clitoral tissue (the authors argued for renaming them as clitoral bulbs). The erectile tissue sizes obtained from their dissections are proposed as being much greater than previously illustrated in various anatomical textbooks. Unfortunately, the actual volume of the clitoral tissue was not quantified using modern stereological technique. An interesting dichotomy arising from the data of Huffman (1969) and of O'Connell et al. (1998) is that while the former showed that the external clitoris actually grew bigger in size even postmenopausally, the latter argued that the internal clitoral tissue is reduced. This suggests that there may be differential preservation of the tissue's androgen receptors in the different structures of the organ.

Because of the long history of identification of the clitoris it is often assumed that ignorance of its function cannot exist in societies even if individuals are ignorant. Herdt (1981), however, reported that for the Sambia people in the Highlands of New Guinea, the clitoris (lakandiku) is accorded no function and no importance is attached to it. Sambian men never mention it publicly. The lack of interest by males in the clitoris is complemented by denials of female orgasm (imbimboogu).

*Clitoral excision.* We know from reports on clitoral excision for cancer (Verkauf, 1975) and on circumcised African females that women can still have sexual desire and conceive without their clitorises. Verkauf (1975) states that 'in those uncommon

instances when clitoridectomy is necessary, available information indicates no decrease in sexual functioning. Women who have experienced orgasm seem to retain this capacity following clitoridectomy.' Megafu (1983) asked a series of questions to 500 female Nigerian Ibos, 340 of whom had been ritually circumcised between the first few weeks of life and puberty. In most cases the whole or part of their clitoris was cut away but their labia minora were left intact. Many of the circumcised women still claimed that they experienced orgasm during coitus (59%) but this compared to a significantly higher figure of 69% in the uncircumcised. Interestingly, the labia minora (35% v 19%) and the breasts (23% v 19%) of the circumcised women became significantly more sensitive than these erotogenic sites in the uncircumcised. The incidence of premarital coitus (its reduction being a traditional reason for circumcising women (Taylor, 1987) was actually unaffected by the circumcision! The conclusion of the author was that the 'sexual urge...is not impaired by the removal of the clitoris' (p. 57). Lightfoot-Klein (1989) interviewed circumcised (removal of clitoris and or/labia) Sudanese women who claimed to still desire sex, enjoy it and even reach orgasm. Yet, in a previous study of 651 Egyptian women who had been circumcised during childhood (Koraim & Ammar, 1965), masturbation was claimed to be less frequent than that observed by Kinsey, Pomeroy, Martin, and Gebhard (1953) in girls who were uncircumcised and it was stated to have a definite effect in reducing the capacity of women to reach their peak of sexual pleasure (orgasm) and a lesser effect on their sexual desire. In the light of the extensive internal clitoral tissue that will still be left even after external clitoridectomy it is not too difficult to expect that numbers of women will still have some erotic feeling left during sexual arousal and coitus from the pressure stimulation of this tissue.

## The Exogenous Activated Desire Phase – Positive Feedback

The other possible position for a desire phase is at the initiation of and during the excitation phase (Figure 1C). In this situation it is relatively easy to see how sexual desire is activated in the female by any or all of the sites shown in Table 1. Stimulation of these erotic friction or pressure sites leads to increased afferent impulse traffic to the brain creating sexual arousal which then activates the desire (unless inhibited) for more such sexual arousal. This is the classic 'positive feedback' mechanism with its end-point culminating in the orgasmic discharge. Unlike the male, however, such a discharge does not necessarily inhibit sexual desire as numerous studies have documented that the human female can experience multiple (serial) orgasms (see Levin (1981) for references).

## Is the Expression of the Human Female Orgasm Identical no Matter how Initiated?

One of the apparent major conclusions arising from the studies of Masters and Johnson (1966) was their powerful definitive concept that 'the fundamental physiology of

the orgasmic response remains the same whether the mode of stimulation remains heterosexual, or artificial coition or automanipulative stimulation of the clitoral area, the breast or any other selected erogenous zone' (p. 132). Thus, according to Masters and Johnson, whatever the area of the female body that is stimulated to create an orgasm, an identical pattern of non-genital and genital activation occurs. In the case of the genito-pelvic musculature, identical uterine and vaginal orgasmic contractions would be induced whatever and wherever the stimulus was applied. This unitary concept of orgasm was in direct contrast to numerous published anecdotal reports of differences of orgasm experienced by women patients of psychoanalysts and therapists when induced by vaginal coitus or by clitoral stimulation. While Freud (1905/1953) is usually credited to be the first to differentiate the nature of the orgasms created by the two modes of stimulation in his clitoral-vaginal transfer theory (clitoral stimulated orgasm being less 'mature' than that induced from the vagina) in reality he did not actually distinguish physiologically between the postulated types. It was his followers who developed the two orgasm concept. One of his school, the psychoanalyst Sylvia Payne (1935), went even further and postulated a third 'blended' or 'merged' orgasm. In her description 'A study of the physical reveals the fact that the orgasm of the vagina which may include muscular contractions of the uterus can be distinguished from orgasm associated with clitoral eroticism. One may and I think should be merged into the other but they are frequently easily distinguishable. Vaginal orgasm has a sucking characteristic, and in some cases the uterus may definitely retract up slightly into the pelvis. Clitoral orgasm is a discharging orgasm and is more like the male orgasm'.

Lowen (1966) summarised the differences thus 'the vaginal orgasm is experienced in the depth of the body, the clitoral reaction is limited to the surface. From what (women patients) say, it appears that only the vaginal orgasm produces the feeling of fulfilment, complete release and satisfaction. In my years of clinical experience, I never heard a woman assert anything to the contrary'. Numerous other psychoanalysts of the Freudian school supported the concept from reports of their patients of differences in the type orgasmic response. Two outstanding features arise from this extensive literature. First the reports were practically exclusively obtained from patients who had clearly gone to the psychoanalysts and secondly, there were no 'controls' from subjects who did not visit psychoanalysts and secondly, there were no experimental observations or measurements to confirm the reports.

Singer (1973), a philosopher, in an analysis of the literature of the various types of female orgastic responses that were proposed before and after the studies of Masters and Johnson, followed Payne (1935) in characterising three types of orgasm:

1. a 'vulval orgasm' showing involuntary, rhythmic contractions of the orgasmic platform (outer third of the vagina) that could be activated by either clitoral or coital stimulation,

2. a 'uterine orgasm' without any contractions of the orgasmic platform but always accompanied by apnoea and a gasping breathing occurring from coitus alone due largely to the 'pleasurable effects of uterine and visceral buffeting stimulating the peritoneum by the thrusting penis through its repeated cervix-uterine contact', 3. a 'blended' orgasm combining elements of the vulval and uterine orgasm and necessitating intromission to bring it about. Repetitive apnoea always appears to occur with this type occurring for about 5 seconds at a time in moments before the climax.

Singer's (1973) evidence for this typology relied exclusively on the descriptions of different orgasms by the novelist Dorothy Lessing (1962), the uterine orgasm, and on the bedroom recordings of sexual arousal by the husband and wife team of Fox and Fox (1969), the blended orgasm.

Recent observations on what really happens in the female genital tract during coitus (Levin, 1998) are highly relevant to these theoretical formulations of Singer. Riley, Lees, and Riley (1992) studied the disposition of the penis inside the vagina during coital thrusting using self scanning ultrasound imaging in ten women. There was a high incidence of penile thrusting reaching the anterior vaginal wall in both parous and nulliparous subjects and the indentation of the base of the bladder was as much as 4 cm deep. In the missionary position, the direct impact of the penis on the posterior vaginal wall or on the cervix was not observed. Penile-cervical contact only occurred in the rear entry 'doggie' position or the rear entry sideways position. This imaging strongly suggests that cervical jostling creating orgasms in the missionary position (by far the most common coital position) is very unlikely.

Recently, however, two sets of static MRI (magnetic resonance imaging) pictures of human face-to-face coitus in the restraining diameter of the apparatus (30 inches / 76 cm) have been published. In one (Weijmar Schultz, van Andel, Sabelis, & Mooyart, 1999) no mention is made of penile-cervical contact in the original paper but in a letter of clarification to the editor (published on the British Medical Journal's website) Weijmar Schultz and van Andel state 'Our women volunteers in experiments 1,2,10-13 mention penile contact with the cervix which could be confirmed by the images'. (The women had full bladders as did those in the previous ultrasound study). In the other static MRI scan of coitus in a woman with an empty bladder, the penis is shown in contact with the anterior cul-de-sac pushing the bladder forwards and upwards (Hatzichristou, 2000). No mention is made of penile-cervical contact. Clearly we are at last beginning to obtain actual images of coitus that should help to answer the penile-cervical contact controversy.

#### **G-spot Stimulation and the Orgasmic Response**

In a completely overlooked article on the activation of human female orgasm, Grafenberg (1950) described how, on clinically examining female subjects, he could greatly arouse them sexually by digital stroking of the upper or anterior wall of the vagina along the course of the urethra but especially in the region where the base of the bladder was positioned. Moreover, in a number of women, this area swelled up on being stimulated to about the size of a kidney bean. The area was re-discovered some years later by Perry and Whipple (1981) and named the G-spot in honour of Grafenberg (Ladas, Whipple, & Perry, 1982). Subsequently, a number of studies were published confirming its presence (see Zaviacic & Whipple, 1993) but others could not locate a definite 'spot' and criticised the concept. Rather than a punctate distribution it was described as a general excitable area running along the whole length of the urethra embedded in the anterior vaginal wall (Hoch, 1986). Hoch reported that when the anterior wall of the vagina was stimulated manually the sexual arousal was almost immediate. Alzate and Londono (1984), working with prostitutes as his subjects, reported their erotic sensitivity located on the upper anterior wall of the vagina but it was in closer relationship to the bladder rather than the urethra. Lenck, Vanneuville, Monnet, and Harmand (1992) localised the underlying structure at the site of the anterior vaginal wall that gave pleasant sensations to the urethral sphincter using ultrasound imaging in the living subjects and dissection of cadavers.

Bearing in mind the recent study on the internal clitoral tissue surrounding the urethra (O'Connell et al., 1998), it may well be that Alzate and Londono (1984), and Hoch (1986), and even Lenck et al. (1992) stimulated this tissue. While we know that friction on the glans and shaft of the clitoris creates excitement, we do not know whether massaging the internal engorged tissue creates the same pleasure. It is clear, however, that in any of the studies where pressure stroking of the anterior vaginal wall occurs, the massage will not only stimulate the G-spot and the underlying tissue complex of Halban's fascia (Minh, Smajda, & Herve de Sigalony, 1979) but also the internal clitoral tissue and stretch the short, female urethra. All these tissues have potential erotogenic activity (Levin, 1992). More recently Chua Chee Ann (1997) has reported that digital stimulation of the inner half of the anterior vaginal wall (an area that he calls the Anterior Fornix Erogenous Zone) lasting 10-15 minutes brings about strong erotic sensations up to orgasmic release in 63% of his clinic subjects (N=271). He interprets this stimulation as being only of a local reflex type with no central expression but there is no evidence for this conclusion at present. Clear-cut distinctions between what stimulated tissue triggers off the 'vaginal anterior wall-activated orgasm' are now much more difficult to make. In reality, it would be more accurate to state that pressure stimulation of the vaginal anterior wall by the penis during active coitus is unlikely to specifically activate only the G-spot. In the context of the complexity of the 'vaginal' erotogenic tissues stimulated by coitus it is salutary to recall the prescient comment of Kinsey, Pomeroy, Martin, and Gebhard (1953) that 'many females, and perhaps the majority of them, find that when coitus involves deep vaginal penetrations, they secure a type of satisfaction which differs from that provided by the stimulation of the labia or clitoris alone. In view of the evidence that the walls of the vagina are ordinarily insensitive, it is obvious that the satisfactions obtained must depend on some mechanism that lies outside of the vaginal walls themselves'. This important aspect of penile coital activity was strangely ignored by Masters and Johnson and was dismissed as patriarchal by feminist apologists.

## Sexual Desire and Pharmacoactive Substances

A wide range of pharmacoactive agents have been reported to influence sexual functioning in both humans and animals usually attributed to their adrenergic, cholinergic, dopaminergic and more recently their serotonergic activity (Riley, Riley, & Brown, 1986; Segraves, 1988b; Meston & Gorzalka, 1992: Kellet, 1993; Melis & Argolis, 1995). While the animal literature (usually rat work) on pharmacoactive agents and sexual activity has many well-controlled experimental studies with acceptable conclusions the human literature is limited, is confusing and is full of single case reports, anecdotal reports and uncontrolled or poorly controlled experiments. From this welter of studies few useful fundamental facts about the role of neuro-transmitters and modulators of human female central sexual mechanisms are available (Bancroft, 1984).

## Vasoactive Agents and Clitoral Haemodynamics

A completely different aspect of genital drug influences has come about from the use of an orally effective phosphodiesterase V inhibitor (Sildenafil) to enhance the local vasodilatation of penile erectile tissues (Boollel, Gepi-Attee, Gingell & Allen, 1996). It does this by increasing the cellular concentration of cyclic GMP by inhibiting its breakdown via the enzyme phosphodiesterase V so that when sexual arousal occurs and nitric oxide (NO) is liberated at the cavernosal smooth muscle sites, its relaxing effect is greatly potentiated and the engorgement of the penis with blood is facilitated. It is one of the most successful oral treatments of erectile dysfunction (Rosen, 1998). It does not create an erection until sexual stimulation occurs nor does it stimulate male sexual desire *per se*. However, as it permits the male to undertake sexual activity with the knowledge that the erection to be induced will almost surely be easier to attain and subsequently be well maintained, a psychological component of taking the drug which will enhance sexual functionality and thus likely sexual desire. This could be so even when 'normal' males take the drug.

No controlled, scientific studies on the effects of Sildenafil on human female arousal and desire have yet been fully published but certain anatomical features of the female genitalia, namely that of the clitoris, will be likely to be involved in its action(s). This organ, as discussed previously, develops from the common tissue anlagen that under the influence of androgen gives rise to the penis. There is sparse nitric oxide synthetase (Nos) activity in the vaginal blood vessels of premenopausal women and none can be located in postmenopausal women (Hoyle, Stones, Robson, Whitley, & Burnstock, 1996). This suggests that NO may not be an important neurotransmitter / neuromodulator in the vagina for creating blood engorgement and lubrication (Levin 1992, 1997). Thus phosphodiesterase V inhibitors would not be very effective in enhancing vaginal blood flow in the premenopausal woman and very ineffective in the postmenopausal. Such constructs are of course speculative, only experimental assessment of vaginal and clitoral haemodynamics in women treated with Sildenafil using quantitative methodology for measuring vaginal blood flow (Levin & Wagner 1997) and clitoral blood velocity (Lavoisier, Aloui, Schmidt, & Watrelot, 1995) will answer these questions. However, a preliminary ultrasonography investigation with the orally effective Sildenafil has reported that it increases the peak systolic velocity of blood in the clitoral artery, interpreted as an increase in blood flow (Berman, 1998) although the diameter of the vessel was not assessed.

A recent study by Burnett, Calvin, Silver, Peppas, and Docimo (1997) reported that in human clitoral tissue obtained at the time of feminizing genitoplasty from 3 girls born with congenital adrenal hyperplasia causing pseudohermaphroditism and one with true hermaphroditism together with that from a normal mature woman, neuronal NOS was observed in nerve bundles and fibres predominantly in corpora cavernosa but also in the glans while endothelial NOS was observed in the vascular and trabecular endothelium with a predominance in the glans. Inducible NOS was not present. Despite the fact that 4 of the tissue samples were from treated patients they had normal sex steroid levels in their plasma and the disposition of the NOS was the same in the patients and in the tissue of the normal woman. The detection of two forms of NOS in clitoral tissue suggests that NO is probably involved in the vasodilatation of the blood vessels of the clitoris and thus by inference phosphodiesterase V inhibitors will be effective in not only increasing clitoral blood flow during sexual arousal but facilitating it even when the stimulus was subthreshold before treatment. This facilitating action is most likely to cause an enhanced feeling of vasocongestion and thus sexual arousal and possibly even that of sexual desire. With all erotogenic tissue (e.g., nipples, penile glans, labia) increasing its blood flow increases its sensitivity to haptic stimuli.

#### Mechanisms for the Discharge of Female Sexual Desire

Now that the major mechanisms for female sexual arousal have been characterised and described and their possible connection to sexual desire examined, those physiological mechanisms that inhibit such desire also need examination. The physiology for the discharge of sexual desire appears different in human males and females. In the sexually aroused mature male, once the ejaculation reflex is activated and the point of 'ejaculatory inevitability' is passed, no voluntary mental activity on the part of the male can prevent the automaticity of the reflex normally accompanied by the orgasmic feelings of intense pleasure. The ejaculation/orgasm usually then creates a feeling of lassitude and a discharge of sexual desire and interest (see model of sexual man as a 'positive feedback-negative feedback, relaxation oscillator' in Levin, 1994). In most males there is then a period, named the refractory period (Masters & Johnson, 1966), when erection is inhibited. The duration of the refractory phase is dependant on the subject's age, on the novelty of subsequent sexual arousal and of the partner. According to Bancroft (1980), it is not proportional to the intensity of orgasmic pleasure. As the ejaculatory and orgasmic mechanisms are independent, males that can prevent ejaculation but still experience orgasm are reported to be able to have repeated, serial orgasms (Robbins & Jensen, 1978). It is claimed that women do not have a consistent identifiable point of 'orgasmic inevitability' that corresponds to that in the male. Moreover, distractions can interrupt the female orgasm unlike males where once the point of inevitability has been passed the ejaculation/orgasm occurs (Masters, Johnson, & Kolodny, 1985).

Because the female does not have an ejaculatory mechanism it is surmised that there is little or no inhibitory feedback from her orgasmic experience, as numerous studies have documented that the human female can experience multiple (serial) orgasms (see Levin (1981) for references). This ability is interpreted to indicate that female sexual arousal and thus sexual desire is not automatically quenched by orgasm. It was this feature that led to the postulation of the sexual insatiability of the human female and the subsequent social sequelae in terms of patriarchal sexual control (Sherfey, 1966). What then terminates sexual arousal/sexual desire in the human female? Why do some women not want more than one orgasm and feel completely satisfied with it? It is unlikely that the supersensitivity of the clitoral glans known to occur post-orgasmic in women (Masters & Johnson, 1966) is the cause of the turn off. Although this supersensitivity can become uncomfortable and even painful it normally only occurs if further mechanical stimulation of the clitoris is post-orgasmically maintained.

While the female clearly does not have an ejaculatory mechanism *per se*, it has been suggested that the contractions of the smooth muscle of the uterus at orgasm are the female equivalent of the contractions of the smooth muscle of the male genital tract at ejaculation.

Returning to the previous discussion of the types of expression of female orgasm by Singer (1973), according to him, that with contractions of the uterus (so-called uterine orgasm) turns off arousal/desire while that without (so-called vulval orgasm) does not. Davidson (1980) discussed these ideas at some length noting that the contractions of the uterus were mediated by the sympathetic system (namely the hypogastric nerve) which corresponds to the same neural control of emission and smooth-muscle mediated part of ejaculation in the male. It was pointed out that it was the centrally activated aspect of these mechanisms that appear to induce the sexual satiation effect not the contraction of the uterine smooth muscle or the smooth muscle ducts of the male. One difficulty in the analysis of the different abilities of the two orgasms to induce female satiation is that uterine contractions appear to occur in both vulval and uterine orgasms. Davidson (1980) overcame this difficulty by proposing that strong uterine contractions are needed for satiation to occur and these are most likely in the uterine orgasm. As yet there is no full laboratory study on female orgasm that has shown that this occurs using physiological recording techniques of the female genitalia that are beyond criticism. However, recordings of the electrical activity of uterine and vaginal muscle activity by Perry and Whipple (1982a) in a single sexually experienced female subject (she acted as a sexual surrogate) produced data that could be interpreted to indicate that clitoral stimulation by the subject created less cervical/uterine electrical activity (4.3  $\mu$ volts) than when her G-spot was stimulated (14  $\mu$ volts). If the sampled voltages can be used as indirect indices of uterine mechanical contractions and that the magnitude of the voltage is an index of the magnitude of the physical muscular contractions, these result would be compatible with stronger uterine contractions initiated from G-spot than from the clitoris. Perry (1998) has now produced graphic data from a 1984 subject recording the two types of orgasmic genitopelvic muscle electrical activity that occurred on the independent stimulation of the anterior vaginal wall (G-spot/urethra) and the clitoris. A ring myograph was fitted into the vaginal introitus of the subject to record contractions of the pelvic striated muscles (bulbocavernosus/ischiocavernosus) while a cervical cap equipped with electrodes that could pick up the electromyographic signals of the contracting uterine smooth muscle was placed over the cervix. Stimulation of her clitoris to induce an orgasm produced little or no signal activity and thus contractions of the uterus but orgasm induced by stimulation of her G-spot produced obvious uterine muscle activity (Figure 2). These single case studies are supportive of the concept of differential

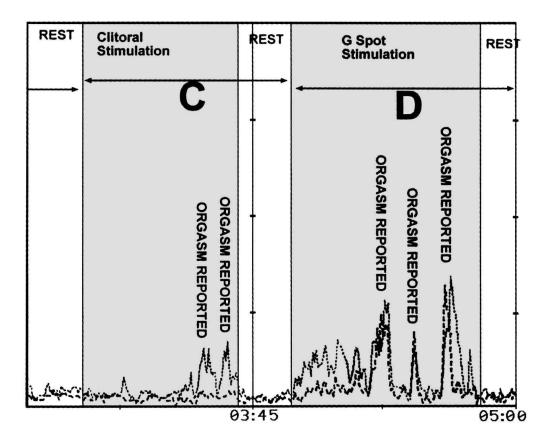


Fig. 2. Simultaneous electromyographical recordings (EMGS) of the contractions of the pubococcygeus (striated) muscle (upper grey trace) compared with those of uterine (smooth) muscle (lower black trace) in a human female sexually aroused to orgasms by either clitoral or anterior vaginal wall (G-spot) stimulation. The pubococcygeus EMG was obtained by an annular ('napkin ring') myograph inserted just inside the vaginal introitus that allowed digital stimulation of the anterior vaginal wall (G-spot area). The uterine muscle EMG was obtained by recording electrodes mounted on the rim of a cap placed over the cervix. The records show that during clitoral stimulation (C) the pubococcygeus activity at orgasm is very much greater than any uterine activity, while during orgasms created from G-spot stimulation (D) both uterine and pubococcygeus activity are recorded. Recordings are by kind permission of John Perry and were taken from his web site.

control of genital muscle function from different stimulation sites but they clearly need to be replicated in other subjects.

# Coitus and Orgasmic Dichotomy

While it is possible to show the dichotomous nature of orgasm in female subjects under laboratory conditions it is likely that during normal coital sexual activity a mixture of the two will occur (the so-called blended orgasm). This will obviously be the case if the clitoris is also stimulated during coitus. Hoch (1986) reported that in those women who stimulated both the anterior vaginal wall and the clitoris at the same time they achieved orgasm very quickly and it was more 'intensive and sexually fulfilling than if obtained by the specific separate stimulation to orgasm of either one of these two areas'. This suggests that there is more than central summation of pleasure from the two genital sites, rather a potentiation of the pleasurable responses.

# How long should Coitus last?

Perhaps a somewhat bizarre heading question but one aspect of the difficulty of locating what turns off sexual desire in women is simply when do women know when sex is over? How long then should coitus last? According to Bose (1937), an Indian psycho-analyst, 'there are males, apparently normal individuals, who could and often do continue for anything from ten minutes to half an hour without stoppage. In such cases the female may experience more than one orgasm in the course of intercourse'. Bose thought that long duration of coitus was not always a sign of disorder sex development but that in most cases, where with uninterrupted movements the duration is habitually above eight minutes, some disorder exists'. He regarded the normal duration of coitus as between one and a half minutes to five minutes. Later estimates by Kinsey, Pomeroy, Martin, and Gebhard (1953) were in the range of 5 minutes. For most men it is relatively easy to know how long the duration of coitus will last, it comes to an end very soon after they have ejaculated! Women, it could be said, even if they are orgasmic, learn from experience that coitus is over for them at the same time! Tiefer (1994) wondered why so much scientific attention is paid to the orgasm, she speculated amusingly that perhaps it 'help(s) people know when sex is over- otherwise they would have to make a mutual decision and that would be too awkward!'. Actually, some 68% of sexually active women (Danish) claimed that they simulated an orgasm in consideration of their partner or to end coitus (Garde & Lunde, 1980) presumably because they were getting tired/bored/or sore from their partner's coital efforts to induce orgasm. Women thus employ the subterfuge of fake orgasms to voluntary control or artificially end 'sexual arousal/ desire'. In a recent study on the handling of various sensory information in relation to sexual activity, Herz and Cahill (1997) found that human females singled out body odour from all other sensory experiences as most able to negatively affect desire. In this respect females were different from males.

# The Natural Loss of Spontaneous Sexual Desire

Human lovers initially have an all-consuming sexual passion for one another, a sexual desire of the highest level and thus at the beginning of the sexual relationship have a high incidence of coital activity. The heat of the sexual desire however inevitably cools down. The intense urgency to make love is gradually replaced by a more measured use of coitus. For some, the sexual desire for the partner fades and can finally become extinct. Once the spontaneous sexual desire has gone it is, as Everaerd and Laan (1995) point out, 'virtually impossible to restore it'. It has been reported that a major reason for low sexual desire in lesbian couples is that they suffer from too much closeness in their relations (Nichols, 1988). Remarkably, such human closeness appears to be inimical to maintain high sexual desire for the partner. This illustrates the concept of some authors that sexual desire is dependent on 'barriers' or 'differences' between people that need to be overcome (Tripp, 1975). If closeness is too great, the two partners fuse into one and there are no differences to overcome. Yet, while 'easy access kills passion and weakens desire. No access at all will also eventually kill passion' (Everaerd & Laan, 1995). Such subtleties and complexities of human sexual desire make it one of the hardest dysfunctions to treat (Leiblum & Rosen, 1988; O'Carroll, 1991). It is not too difficult to see why there is so much current interest in the application of phosphodiesterase V inhibitors and other pharmacological treatments to enhance female sexual arousal. Humans always hope for a magic pill to cure their ills!

# Conclusion

Over the reviewed millennia, the remarkably slow development of mankind's understanding of human sexual arousal mechanisms is clear. Apart from a few early landmark studies, little significant physiological progress occurred until the middle of the twentieth century! Even then most of this increase in knowledge has been about the peripheral functioning of our genital organs, epitomised perhaps by the EPOR schema. The working relationships between the genital organs and the brain and the sexual mechanisms that take place in the 'black box' of the brain, as exemplified by the DEOR model, are only just beginning to be able to be studied in humans (Levin, 1994; Tiihonen, Kuikka, Kupila, Partanen, Vainio, Airaksinen, Eronen, Hallikainen, Paanila, Kinnunen, & Huttunen, 1994; Stoléru, Gregoire, Gerard, Decety, Lafarge, Cinotti, Lavenne, Le Bars, Mazover, Magnin, Spira, & Comar, 1996; Komisaruk, Whipple, Gerdes et al., 1997). Human sexual desire has arguably been the least successful area for such scientific study. Tiefer (1994), who interprets human sexuality from the social constructionist's viewpoint, argues that 'physiology should be the background of a model of sex, not the foreground' and that 'The role of physiology in sex is similar to the role of physiology in dancing. It's there - it is important in some ways, but it is not central to the meaning and the experience of the enterprise'. Notwithstanding the strained dancing analogy, while there are clearly important aspects of sexual desire that are of social construct (historical examples were illustrated in the introduction) and of personality development, the role of its biological substrates should not be marginalised. As E. O. Wilson said 'Biology does not dictate our behaviour but it keeps it on a short leash'.

Although the withering of sexual desire in women cut off from their genitals by complete spinal cord lesions as first reported by Money (1960) does not now appear to be the case for all spinal cord injured females (Komisaruk, Gerdes, & Whipple, 1997) the investigation of the genitopelvic neural afferent input to the brain may still be an important aspect of the maintenance of normal sexual desire. Exactly what messages our genitals send to our brains and how they influence the neurotransmitters and neuromodulators that will be found to be the mediators of sexual desire will be a difficult but fascinating area for study (see the reviews by Komisaruk & Whipple, 1995; Whipple & Komisaruk, 1999). The old, often disparaged, adage that 'anatomy is destiny' has not disappeared, it has become superseded by the newer 'anatomy is destiny, but physiology drives you there'!

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