# SPATIAL ABILITY IN MEN AND WOMEN: REVIEW AND NEW THEORY

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"In matters controversial my perception's rather fine:
I always see both points of view, the one that's wrong, and mine."
from 'Is it really so?' by Dwight J. Ingle, 1976.

**Abstract** — Results from research on spatial abilities are selectively reviewed. Differences are noted in the effect of puberty on spatial ability of men and women, in the development of spatial ability in early- and late-maturing boys and girls, and in the spatial ability of feminine men and masculine women. Abnormalities are described in the spatial ability of men and women with hormonal disturbances. Variations are found in the spatial ability of women during the menstrual cycle. These findings are considered to lack an adequate explanation, so a new theory is proposed to account for them. The theory considers the cerebral level of estrogen to play an essential role in the expressions of spatial ability while testosterone is said to modulate actions of estrogen. The sex specific ontogenetic pattern of plasma hormone values and the loci of their biological action are outlined, and central mechanisms for hormone-brain-behavior relations are discussed. The theory is used to account for recent findings on spatial ability of women and men. Several implications of the theory are considered, and experiments needed to test the theory directly are discussed.

## INTRODUCTION

Spatial ability is a complex trait. It plays a role in a variety of perceptual, cognitive and perceptual-motor tasks and seems related to certain personality traits. Competence in mathematics, geometry, physics, mechanics, engineering and architecture requires spatial ability. Achievement in science in general, non-verbal aspects of most standard IQ tests, tests of psychological differentiation (field-dependence), direction finding, map reading and orientation in space also depend heavily on spatial ability (Bennett *et al.*, 1974; Crandall and Sinkeldam, 1964; French, 1951; Goodenough and Karp, 1961; Gray, 1979; Guildford *et al.*, 1951; Hills, 1957; Horn, 1976; Hyde *et al.*, 1975; McGee, 1979; Saad and Storer, 1960; Sherman, 1967, 1974; Smith, 1948, 1964; Sweeney, 1953; Waber, 1977; Werdelin, 1961; Vernon, 1950). Clearly, spatial ability is an important and pervasive higher mental skill.

Spatial ability has been proposed to have two major components, namely orientation and visualization, and a number of minor components (McGee, 1979; Smith, 1964). Spatial orientation refers to the ability to comprehend the arrangement of elements within a visual stimulus pattern, the ability to deal with changing orientation in which a spatial configuration is presented and the ability to determine spatial orientation with respect to one's body. Spatial

visualization refers to the ability to mentally manipulate, rotate, twist or invert a visual stimulus. Recognition, retention and recall are involved in visualization in 2- and 3-dimensions. Harris (1981) noted, however, that consensus is still lacking on the exact meaning of visualization and orientation, and that the task of identifying the critical components of various 'spatial' tests is still part guesswork. Eliot (1980) pointed to the persistently high correlations found between visualization and orientation tasks, and suggested that perhaps we have not yet reached the stage where we are ready to generate fully logical classifications on the basis of available factorial research. Smith (1964) was worried by the observation that different populations and different conditions of test-administration can produce different factor-loadings in the same spatial tests, and preferred to use the term 'spatial ability' to represent a complex family of abilities with unknown interrelationships. Finally, Liben (1981) regretted that space, like time, is a concept that we seem to understand intuitively, but that becomes uncomfortably elusive when we try to define it. The best we can do in this bewildering situation is, perhaps, to realize that lack of exact definitions *a priori* should never prevent the search for new knowledge (Popper, 1973). Therefore, we may make the assumption — based upon at least superficial similarities among various tasks — that there are certain discrete skills common to all the different tasks that have been called 'spatial' (Harris, 1981), and further to all those tests or subscores of tests which have been found to consistently correlate significantly with spatial ability tasks.

A striking finding is that men are usually found to be better than women in tasks based primarily on spatial ability (Anastasi, 1958; Garai and Sheinfeld, 1968; Maccoby and Jacklin, 1974; Smith, 1964; Tyler, 1965). Adult men typically obtain higher scores than adult women on, for example, Porteus' Mazes, Money's Road-Map Test, Piaget's Perspectives, Water Level Tasks, Geometric Forms and House Plans Tasks, Rod-and-Frame Test, Embedded Figures Test, Tilting Room Tilting Chair, Mental Rotations, WAIS Analytic Triad. Evidence for the role of spatial abilities in tasks comes from the fact that removal of the spatial component by the use of statistical procedures eliminates the superiority otherwise shown by men (Fennema and Sherman, 1977; Geiringer and Hyde, 1976; Hyde *et al.*, 1975). The notion that high spatial ability, is responsible for the superiority of men in these tasks also receives some support from the temporal relation between the development of sex differences in spatial ability and sex differences in task performance. Thus, a male lead in spatial ability is typically found around puberty at which time the male superiority in the various tasks appears (e.g. Maccoby and Jacklin, 1974). Of course, such observations do not rule out the contribution of other variables such as motivation, experience and cultural stereotypes, but the development of a sex difference in spatial ability is most probably one of the more powerful variables behind male superiority in such tasks. It is, therefore,

of interest to study the etiology of the sex difference in spatial ability. A number of social, biological, and interaction theories have been advanced to explain the findings. Unfortunately, most of these theories run into a variety of problems (see later), and it is likely that new explanations are needed to account fully for the etiology of sex differences in spatial ability.

The purpose of the present review is to consider studies on spatial ability in normal and abnormal men and women. The findings suggest a new theory to account for individual differences in spatial ability in men and women. The theory is described and experiments needed to test the theory directly are mentioned.

## REVIEW

### Spatial Ability in Prepubertal Girls and Boys

Coates (1974a, 1974b) and Keogh (1971) found preschool girls to be superior to preschool boys in spatial ability, at least at certain ages. In addition, superiority of young girls to boys in spatial ability was verified using the WPPSI block design subtest (Kogan, 1976). On the other hand, several studies found prepubertal boys to do better than girls (Fairweather and Butterworth, 1977; Kohen-Raz, 1977; Mercer and Smith, 1972; Nisbet and Illsley, 1963; Roberts, 1972; Yule et al., 1969; and others). Most other studies indicated, however, that prepubertal girls and boys are much alike in most spatial ability tasks (Maccoby and Jacklin, 1974). A comparative study of the development of various aspects of prepubertal spatial abilities is called for, as perhaps not all spatial abilities develop at the same rate.

### Spatial Ability in Early and Late Maturing Boys and Girls

Nisbet and Illsley (1963) found that early maturers were mentally precocious during late childhood and at adolescence, but that their mental superiority tended to disappear again during post-adolescence. Douglas and Ross (1964) also noted a superiority of early maturers that continued at least up to the age of 15 years. In a series of studies, Broverman and coworkers demonstrated low spatial ability (strong automatization) in individuals that had matured early and high spatial ability (weak automatization) in late maturers (see for example Broverman et al., 1964). Waber (1976, 1977a) found that late maturers showed better spatial ability than did early maturers. In addition, Waber demonstrated that late maturers showed stronger ear advantage on a dichotic listening task (often taken to indicate greater

hemispheric lateralization) than did early maturers of the same sex and age, and called attention to similarities between early maturers and field dependent persons and between late maturers and field independent persons (1977b). Kohen-Raz (1977) also studied early and late maturers and observed that before puberty early maturing girls showed superior mental ability while after menarche the relation tended to invert so that late maturing girls showed mental superiority. Applying Raven's progressive matrices, Kohen-Raz found that upper middle class *taller* girls (i.e. early maturers) were superior during fifth and sixth grade while this trend disappeared by the eighth grade. Among boys, only the taller eighth graders obtained significantly higher Raven Scores. This pattern was, however, not found in lower middle class children. Neither could Petersen (1976) and Herbst and Petersen (1979) replicate the relationship between maturation and spatial ability in normals, nor could Rovet (1979) confirm it in clinical groups with precocious or delayed pubertal development. Nyborg and Nielsen (1979) noted, however, that girls with Turner's syndrome and pubertal delay have much lower spatial ability than have their sisters. Short-term cyclic estrogen treatment speeded up their pubertal and perhaps also spatial ability development (Nyborg and Nielsen, 1981a).

Interpretation of these results is complicated by the fact that the criteria for maturation differed from study to study, because early and later maturers were not always tested at the same age in the various studies, and because some studies used extreme groups. Furthermore the terms 'high' and 'low' for one group are only relative to the scores of the opposite maturational group. Also, some of the studies applied standard intelligence tests with considerable load on verbal skills. Waber (1976, 1977a) was, however, unable to observe a verbal ability-maturation relationship. Longitudinal studies are needed in which the degree of maturation of each single individual is related to his or her spatial scores. Nevertheless, some trends can be seen in the data. In general, early maturing persons appear to obtain a slightly elevated score on tests containing a spatial ability component, but tend to decline in early post-adolescence. Late maturers tend to score lower than early maturers at first, but after puberty late maturers take the lead.

Several explanations have been offered for the pubertal decline in spatial ability of the early maturer. Kohen-Raz (1977) explained the disappearance of the mental superiority of early maturing girls by referring to their greater emotional sensitivity and instability, while Waber (1977a) speculated that puberty might inhibit the maturational lateralization process or lead to a cerebral reorganization (Waber, 1979). Vandenberg and Kuse (1979) noted that the relation between physical and mental development is neither simple nor well understood and further that a purely maturational theory of the observed sex differences in spatial ability fails to explain the constancy of the difference when the growth period is over.

## *Spatial Ability in Normal Women*

Spatial ability is a continuous trait and it is worth remembering that men with very low spatial ability and women with very high spatial ability can be found quite easily in most populations. The variation in spatial ability within sex is tremendous and the overlap between sexes is considerable. Nevertheless, the lower average spatial ability in adult women than in adult men must be acknowledged and can be expected to impair the performance of women in tasks containing a spatial component. What is more, selection for certain occupations is based on the upper percentiles of the spatial ability distribution, and there men are clearly over-represented. Helson (1971) and Smith (1964) thus speculated that low spatial ability in women explained the rarity of creative mathematicians. Finn *et al.* (1979) showed that the sex gap disfavouring women in science achievement was larger in the physical sciences than in biology, perhaps because physics draws more heavily on spatial ability than does biology. Recently Kelly (1978) thoroughly documented the generality of a female disadvantage in science achievement comparing fourteen-year-old pupils in fourteen different countries. The female disadvantage was uniform across cultures and sub-cultures, indicating that specific cultural expectations and pressures had little influence on the development of this sex difference.

One source of the relatively low spatial ability of adult women may be that the development of these skills levels off in early adolescence in girls, while it continues in boys. It might also be, however, that while most boys reach an asymptote in spatial ability around or shortly after puberty, most girls show a decline during puberty and their decline is larger than that seen in boys. Witkin *et al.* (1967) obtained evidence in support of the latter idea. They found a postpubertal decline in spatial ability in girls, using the Rod-and-Frame Test in a cross-sectional study. Witkin *et al.* discussed the possibility of a 'return to field dependence' in girls, but concluded that their observation might have been due to artifacts in the selection of their subjects. However, in a replication of the study by Witkin *et al.*, Nyborg (Note 1) observed a similar postpubertal decline in spatial ability in girls which could not be ascribed to selection artifacts. Such a decline has been referred to as a tendency to 'return to systematic optical-dominated strategies' (Nyborg and Nielsen, 1979). An analogous pubertal depression of Embedded-Figures Test scores and of Money's Road-Map Test scores has been observed in girls (Nyborg, unpublished observation). Much other evidence also supports the notion of a greater decline in spatial ability at puberty in girls than in boys. For example, a linear decline in a visual discrimination task occurred in girls after the age of 16 (Noble *et al.*, 1964), and rotary pursuit performance began to deteriorate at about the age of 15 years in girls (Ammons *et al.*, 1955). Sandström and Lundberg noted that women show an absolute decline in performance of

spatial localization tasks compared to girls. Droege (1967) administered a spatial ability measure (General Aptitude Test Battery) and found 17 year-old girls to be outscored by boys by nearly half a standard deviation. Wolf (1971) found that girls began to be at a disadvantage to males in a new non-verbal visual perceptual test for spatial ability just about adolescence. The observations by Wolf and by Droege could, however, also be seen as a relative acceleration in boys. Maccoby and Jacklin (1974) noted that visual-spatial tasks have a developmental course in which the female disadvantage emerges in early adolescence. The International Association for the Evaluation of Educational Achievement applied identical tests for mathematical achievement on a nation-wide basis in twenty-one countries. Comber and Keeves (1973) summarized the results on measures of ability and (science) achievement, and found that ten-year old boys outscore girls by only one-fourth of a standard deviation, while at the age of fourteen, girls were behind boys by one-half of a standard deviation. The difference between boys and girls increased to nearly a full standard deviation for students in their final year of secondary school. Macroff (1975) observed that girls' achievement test scores in mathematics began to decline at the age of thirteen, while Ross and Simpson (1971) found mathematical ability to decline in girls between the ages of 11 and 15 years. Stafford (1972) could observe no prepubertal sex difference in quantitative reasoning but found that boys took the lead after age twelve or thirteen. Bayley and Oden (1955), Kagan et al. (1958), Sontag et al. (1958) and Terman and Oden (1947) all found that after elementary school more girls than boys demonstrated a declining pattern of IQ. Campbell (1976) noted that the IQ of girls declined between seventh and twelfth grades while the IQ of boys increased during that period. Kangas and Bradway (1971) found that girls scoring high prepubertally on IQ tests showed the least gain after puberty. The findings all support the notion that intellectual changes measured by current tests and achievement indices often tend to show a decline or levelling off in pubertal girls, while they tend to increase in boys or to reach an asymptote in boys (Jarvik, 1975). In the present context it is important to note that a female pubertal decline in full IQ seems attributable mainly to a regression in non-verbal components of these tests, in that girls usually begin to outperform boys in at least some of the verbal areas starting at 10–12 years (Maccoby and Jacklin, 1974).

  Unfortunately, most studies on sex differences in development of spatial ability present only group mean scores. As a result, the findings fail to show the course of individual development in spatial ability. Accordingly detailed information on the pubertal decline in spatial ability in girls is almost completely lacking since the approach of averaging data in groups disregards the actual number and kind of persons showing a decline. Thereby a declining tendency within a group might be camouflaged by other persons demonstrating a relative acceleration. To avoid this error, Hindley and Owen

(1979) presented person-specific longitudinal curves for IQ's in the time span between 6 months and 17 years. By this approach they were able to classify the curves into seven categories. They found that some subjects experienced a considerable pubertal decline with more girls than boys showing a decline although the difference was not statistically significant (Hindley, personal communication). Perhaps the difference would reach significance if the spatial components in the test were isolated. Nyborg (Note 1), using a person-specific approach to the Rod-and-Frame Test, found that more girls than boys showed a decline in performance around puberty and that the decline tended to be larger in girls than in boys.

It is to be noted at this point that the degree of sexual maturation of women, judged in terms of behavior and secondary sexual characteristics, apparently is related to spatial ability in a relatively straightforward way. Numerous studies show high "femininity" to be associated with non-enhanced spatial ability and high androgyny (i.e. women ranking high on some masculine attributes in addition to demonstrating the usual feminine attributes) to be associated with enhanced spatial ability (Petersen, 1976; Waber, 1976, 1977a; see also Maccoby and Jacklin, 1974). Bodily measures of degree of femininity and masculinity include anthropometric size estimations, examination of the distribution of pubertal hair and body fat, and estimation of total body water. These measures have shortcomings and are typically obtained in cross-sectional rather than longitudinal studies. As a result, the exact relation between bodily appearance and spatial ability remains to be established with certainty. However, studies performed so far show androgynously appearing women to have higher spatial ability than their more "feminine" looking sisters (Maccoby and Jacklin, 1974).

Women differ considerably with regard to sex role identity and femininity-masculinity. Unfortunately the measurement of these psychological traits suffers not only from the lack of a metric scale such as is available for body measures, but also from conceptual confusion (see Constantinople, 1973). It has been suggested that masculinity-femininity ought to be measured not only on a scale with "masculine" and "feminine" at the extremes (or on two separate scales), but simultaneously on an orthogonal scale with "demasculinized" and "defeminized" at the extremes (Whalen, 1974), or perhaps with the addition of an oblique "demasculinization-defeminization" scale (Reinisch, 1976). Whatever the proper solution may be, it is noteworthy that the relation between sex role traits and spatial ability is very similar to that between bodily appearance and spatial ability. Thus, women with strongly feminine sex role performance show non-enhanced spatial ability, while women with a stronger than average androgynous sex role performance show enhanced spatial ability (Ferguson and Maccoby, 1966; Jamison and Signorella, 1980; Maccoby and Jacklin, 1974; Signorella and Jamison, 1978; Welsh and Baucom, 1977).

Another important aspect of the etiology of sex differences in the development of spatial ability concerns effects of menstruation. Anderson (1972) and Klaiber and coworkers (1974) found spatial performance to vary with the menstrual cycle, being highest in the low estrogen phase. Dor-Shav (1976) noted that embedded figures task performance was optimal when estrogen values were decreasing and at their lowest. Wickham (1958) observed scores in a practical mechanical test to be higher during the menstrual period when estrogen production is practically nil, and Wuttke *et al.* (1975) found scores on mental arithmetic tasks always to be higher during periods of low estradiol levels. Wuttke *et al.* also noted that calculation times increased and showed no cyclical changes when their subjects were treated with combined norgestrel/ethinyl-estradiol contraceptives. Diamond *et al.* (1972) noted that visual sensitivity increased with increasing estrogen levels, reached an optimum at midcycle when estrogen is highest, and declined abruptly at the onset of menstruation when estrogen production is practically stopped. Vogel *et al.* (1971) observed a relation between blood estrogen levels and EEG driving responses. Sommer (1972) on the other hand found no relation between hormones and cognitive performance, and suggested subsequently (1973) that previous observations could be ascribed to socio-cultural expectations about menstruating women. A more balanced view of biological and socio-cultural factors was taken in a review by Dan (1979) who noted that women might be more sensitive to some stimuli at one point and to other stimuli at other times as a function of menstrual phases. Such a cyclic change might be reflected in the observation that "within-pair" variance of psychological tests among female twins is greater than among male twins (Vandenberg *et al.*, 1962).

The observation that the sex differences in spatial ability scores reach a zenith at the time when the "female" and "male" sex hormone concentrations in plasma are maximally different between women and men; the finding that sex-specific bodily differentiation is correlated to spatial ability differentiation; and the notion that spatial performance apparently varies with hormonal changes during the menstrual period suggests a role for sex hormones in spatial ability, but certainly proves nothing. Direct tests of causal relations between the level of sex hormones in humans and their level of spatial ability are required in order to provide unequivocal proof for such a hypothesis. But ethical considerations preclude experiments in which large numbers of normal individuals are randomly assigned to various experimental groups and exposed in a double-blind design to sufficiently large amounts of various sex hormones in order to provide solid information about the possible effects of hormones on spatial ability. Therefore, a methodologically less rigorous approach may be used to test the hypothesis. It involves the study of spatial ability in groups of individuals with abnormal levels of sex hormones due to either endogenous or exogenous reasons. The following section reviews

studies of spatial ability in such groups of women. The main question to be answered is whether the trend observed between sex hormone levels and spatial ability in normal women are also found to occur systematically in women with sex hormone abnormalities.

## Spatial Ability in Abnormal Women

Spatial ability has been studied intensively in women with Turner's syndrome (for review, see Nielsen *et al.*, 1977). These women are genetically and hormonally abnormal. Women with Turner's syndrome lack one sex chromosome partly or completely in all or some of their cells. Phenotypically these women are short in stature. Their gender role identity is definitely feminine. They wear typically more delicate and more "feminine" types of jewellery as compared to their sisters and also dress themselves in a distinctively more feminine way. They have repeatedly been found to be extremely low in spatial ability, and a number of hypotheses have been set forth to explain this. Recently, Nyborg and Nielsen (1981*a*) examined a group of women with Turner's syndrome that received short-term cyclic treatment with the "female" sex hormone estrogen (for between 3 months and 2 years; mean 1 year 2 months, SD 11 months) and found them to have spatial ability identical to that of their matched sisters. On the other hand, long-term estrogen-treated women with Turner's syndrome (over 4 years of cyclic hormone treatment; mean 8 years 1 month, SD 3 years 6 months) scored as low on spatial ability tasks as did untreated women with Turner's syndrome. These results suggest the potential importance of variation in plasma estrogen concentration in the expression of spatial ability.

Studies of women with the adrenogenital syndrome may provide further hints about the effect of sex hormones on spatial ability. The adrenogenital syndrome (AGS or congenital adrenal hyperplasia) is an autosomal recessive disorder in which the adrenal glands produce abnormally high amounts of androgen, beginning around the third month of foetal life. When discovering AGS, girls are usually treated with cortisone to adjust them to a nearly normal level of plasma androgen. Considerable effort has been devoted to the study of cognitive development in such females, and the results obtained so far are controversial. AGS girls show higher overall IQ and greater school achievement than unrelated controls, although their scores are not superior to those of unaffected siblings or their parents (Baker and Ehrhardt, 1974; Erhardt *et al.*, 1968; Ehrhardt and Money, 1967; Money and Lewis, 1966). AGS girls tended also to outperform their unaffected siblings on Cohen's perceptual factors in the Wechsler tests, although the difference was not significant. Perlman (1973) noted high "non-verbal evaluative" abilities in AGS girls. Baker and Ehrhardt (1974), on the other hand, found the

adrenogenital syndrome to affect neither verbal-performance IQ nor Primary Mental Ability Spatial Test. They concluded that the prenatal exposure to androgen in this syndrome does not appear to influence later cognitive development. Unfortunately, age is not corrected for in most of these studies. Accordingly, conclusions about lack of effect of surplus androgen on later cognitive development was often made on prepubertal children, in whom a sex difference in spatial ability usually is not found anyway. It is also to be noted that comparisons of performance IQ between girls exposed to high androgen levels and normal girls showed an elevation of performance IQ in androgen-exposed girls.

The spatial ability of daughters born to women treated with progesterone during pregnancy may show relations between sex hormones and performance on spatial tasks. At least some progesterones are known to have androgen-like properties, and daughters born to women given these drugs are sometimes masculinized at birth, a condition that is amenable to surgical correction. The daughters have been found to obtain higher IQ scores than unrelated controls, to be more likely to pass university entrance examinations, and to be especially keen in science subjects (Dalton, 1957, 1968, 1976). Furthermore, a relation between the dose of progesterone administered to mothers and the spatial and mechanical ability of the daughters was observed, in that earlier and higher progesterone medication produced a greater elevating effect on these abilities. Zussman *et al.* (1975) further noted significant relationships between prenatal progesterone treatment and spatial ability. Lynch and Mychalkiw (1978) claimed on the other hand that Dalton used inappropriate statistical procedures, and concluded on the basis of a re-analysis of her data, that the postulated effects of prenatal progesterone on later cognitive development were non-existent. Lynch *et al.* (1978) then studied the prenatally exposed progesterone children by a number of tests including spatial tasks and observed no differences between progesterone children and normal controls. Reinisch (1977) and Reinisch and Karow (1977) furthermore, failed to find differences between any of the Wechsler tasks comparing untreated siblings and female off-spring of women treated with a predominance of synthetic progestin during gestation, or estrogenic hormones including diethylstilbestrol. The last three studies have been taken to indicate that prenatal progesterone exposure has no effect on later cognitive development. This may be incorrect, however, because the age factor again was not taken into account. As mentioned previously sexual differentiation in spatial ability typically appears consistently only in the post-pubertal period. But the age range of the progesterone children in the study by Lynch *et al.* (1978) was 14.9–17.1 years with more than half of the children below 16 years of age, and the usual postpubertal sex differences in spatial ability could not be observed between the seven girls and the four boys who served as their control group (mean age 16.1 ± .8 years). In the studies by Reinisch, the mean age of

the children was less than thirteen years. It would be interesting to re-examine the progesterone children mentioned in the negative reports at a later age. In the meantime, it still seems reasonable to suppose that prenatal progesterone exposure has postpubertal effects on spatial ability development. An observation supporting this idea was made by Zussman *et al.* (1975) who studied spatial ability in 16–19 year old progesterone children; they found significant relationships between prenatal treatment and spatial ability in this age range.

The androgen-insensitive syndrome is another condition in which a relation between sex hormones and spatial ability is to be expected. The syndrome refers to individuals born with cells that do not respond properly to androgens due to an X-linked recessive error. These individuals have no internal sex organs and usually lack pubic hair, but develop otherwise as normally appearing women, even if they have the male karyotype. Androgen-insensitive persons have the typical "female" pattern of higher verbal than performance IQ scores (Bock and Kolakowski, 1973; Masica *et al.* 1969; Money and Ehrhardt, 1972; Money *et al.*, 1968; Money and Ogunro, 1974; Spellacy *et al.*, 1965) suggesting that the male pattern of superior visual-perceptual organization normally expressed directly or indirectly by the 46,XY male karyotype is totally lacking in patients with the complete form of testicular feminization (Masica *et al.*, 1969, p. 41). Further evidence for a role of androgens in spatial ability comes from data on postpubertal women exposed endogenously (the andrenogenital women) to higher than normal levels of prenatal androgens. In general, these conditions are accompanied by higher than normal non-verbal IQ and increased spatial and mechanical ability. On the other hand, individuals insensitive to androgens have lower performance than verbal IQ and tend to have low spatial ability. These findings are difficult to interpret, however, for reasons already mentioned.

### Spatial Ability in Adult Men

Adult men tend to score higher than women on spatial tasks (Maccoby and Jacklin, 1974). But as was the case with females, the variability within male groups is tremendous, and the overlap between male and female distributions points to serious difficulties in unqualified use of sex as *the* paramount variable in spatial ability research. As with women, a relatively unambiguous relation between spatial ability and maturation of the sexual characteristics has been found in men. However, it may seem surprising that "feminine" looking normal men, i.e. androgynous men, tended to score *higher* than "masculine" looking men on spatial tasks (Petersen, 1976; Waber, 1977*a*; see also Maccoby and Jacklin, 1974). With all the pitfalls and shortcomings in the measurement and understanding of sex role traits in mind, one might

nonetheless by analogy expect men with a high self-reported androgyny score to demonstrate enhanced spatial ability and men with a high masculinity score to obtain non-enhanced spatial ability scores. This was, in fact, found in several studies (Maccoby and Jacklin, 1974).

The fact that androgynous men score high on spatial ability tasks creates a paradox from the sex-typing point of view that enhanced spatial ability is a socially defined masculine trait. The relation suggests, on the other hand, that plasma hormone concentrations might be important for the enhancement of spatial ability in men as they seem to be in women. The following section examines this idea further by reviewing studies on spatial ability in hormonally abnormal men.

### Spatial Ability in Abnormal Men

Men with karyotype 47,XYY look undisputably like men, and are often taller than the average man. They are often characterized as impulsive and as having a low tolerance for frustration or anxiety (Nielsen and Christensen, 1974). Nyborg and Nielsen (1981) observed that men with karyotype 47,XYY scored much lower than normal women on spatial ability tasks such as the field dependence indicators (the Rod-and-Frame, Embedded-Figures, and Human Figures Drawing Tasks) and Money's Road-Map Test for direction sense. A massive depression of spatial ability in men with an extra Y-chromosome has not been reported in previous studies but was confirmed in an independent study with matched controls (Theilgaard, personal communication).

Men with Klinefelter's syndrome have karyotype 47,XXY. Phenotypically such men have male genitalia, small testes, feminine fat distribution and sometimes female breast development (see for example, Nielsen, 1969). Nyborg and Nielsen (1981) found spatial ability in men with Klinefelter's syndrome to be lower than the average male level and indiscriminable from the female average.

Protein-deficient men may develop the Kwashiorkor syndrome. Phenotypically this often feminizes the body such that feminine fat distribution and female breast development can be observed in these men. In addition they tend to have an abundance of estrogen. Dawson (1966) found that men with Kwashiorkor syndrome score very much like women, i.e. they were field dependent and thus low in spatial ability. In a study of protein-energy malnutrition and intellectual abilities in teenage Ugandan children, Hoorweg (1976) observed that there was a general impairment of intellectual abilities concomitant to malnutrition, and that reasoning and spatial abilities were the most affected.

Men with the adrenogenital syndrome tend to obtain higher IQ scores than

those achieved by normal unrelated controls (Ehrhardt, 1975; Ehrhardt and Baker, 1974). It is to be recalled that the adrenogenital syndrome results from prenatal exposure to higher than normal male concentrations of androgens. While spatial ability of individuals with the adrenogenital syndrome is elevated relative to unrelated controls but not relative to family members, their verbal score seems to be even more elevated compared to controls.

Other types of prenatal exposure to disturbances in sex hormone balance also appear to influence spatial ability in men. Thus, Reinisch (1977) found that exogenously-induced prenatal exposure to more progestin than estrogen further masculinized male offspring and influence a number of personality traits in a masculine direction, i.e. the young boys were markedly more individualistic, independent and self-assured than siblings not exposed to progestin. Young boys exposed *in utero* to more estrogen than progestin obtained scores typical of girls on Cattell's personality questionnaire, i.e. they were counted as group-oriented, dependent and showing lack of self-sufficiency. No differences were observed on Wechsler subtests scores between the progestin/estrogen, estrogen/progestin, and control groups. It should be remembered that the mean age for the groups in the study by Reinisch ranged from 10.61 to 12.46 years. It would also be interesting to know if hormonally related differences in spatial ability appear shortly after puberty in these groups. A study by Yalom *et al.* (1973) demonstrated the importance of controlling for age in such studies. They studied a group of 6-year-old boys and also a group of 16 years of age, and compared these groups on a number of personality traits and on field dependence as a function of different levels of prenatal estrogen exposure. The prenatal level of estrogen was inferred from the fact that some of the boys had diabetic mothers who received supplemental estrogen treatment to compensate for their abnormal low plasma estrogen due to their diabetic condition (the high estrogen group). Other boys were sons of normal mothers (the intermediate estrogen group). Finally, some diabetic mothers received no hormonal compensation (the low estrogen group). The groups of 6-year-old boys differed only with regard to lower self-assertiveness and athletical ability in the high estrogen group. At 16 years of age, however, a difference in spatial ability was apparent. It was in the expected direction in that the boys exposed to high prenatal estrogen levels obtained the lowest spatial ability scores as measured by the Embedded-Figures Test in addition to being the less aggressive, less assertive, and the less athletic group.

The spatial ability of hormonally abnormal men provides further support for the idea that the expression of spatial ability is related to hormone levels. The relation is, however, not a straightforward one, as abnormal plasma testosterone levels and/or unusual aromatization in 47,XYY men were accompanied by low spatial ability, while low plasma testosterone levels in 47,XXY men also were associated with low spatial ability as in men with

androgen-insensitivity. Apparently, either too high or too low plasma testosterone levels depresses the expression of spatial ability. The observation that adrenogenital boys (and girls) with high testosterone levels show high spatial ability is not necessarily contradictory to this idea, because the parents and unaffected siblings to adrenogenital persons also show elevated IQ's, and this has been interpreted to mean that the general elevation of IQ was not due primarily to an abnormal hormonal condition but rather to unknown factors concomitant to families in which the adrenogenital syndrome is seen (Baker and Ehrhardt, 1974) or to sampling bias (Perlman, 1973; Reinisch, 1977). These problems are presently unsolved, so spatial ability in patients with the adrenogenital syndrome will not be considered in the present context. Nevertheless, the idea that sex hormone level may be of major importance for the expression of spatial ability was suggested strongly enough by the other studies to be taken seriously. The idea that either high or low levels of plasma testosterone might be damaging to the expression of spatial ability provides an explanation for the findings obtained in an experimental testosterone-infusion study by Klaiber *et al.* (1971). However, the observation that high estrogen levels in males with the Kwashiorkor syndrome and in sons of diabetic mothers were related to low spatial ability suggests that estrogen level may be at least as important as testosterone level for the expression of spatial ability.

### Summary of Results of Studies of Spatial Ability in Women and Men

Particular attention was given in the present review to studies providing evidence that seems to oppose traditional theories on spatial abilities. The findings reviewed lead to the following tentative conclusions:

1. Spatial ability in general develops rapidly and equally in both sexes until the time of puberty.

2. Most boys reach an asymptote in spatial ability around puberty while a few show a decline. At puberty, some girls reach an asymptote while the majority decline to a prepubertal level.

3. Regardless of sex, physically early maturing children show relatively higher spatial ability than do late maturing children, but their superiority disappears again. Regardless of sex, physically late maturing children tend first to show relatively lower spatial ability than do early maturing children. Later on, spatial ability in late maturers rises and surpasses that shown by early maturers, and this superiority seems to last.

4. In adulthood there is a slight but significant group mean advantage in spatial ability in favour of men.

5. Spatial ability in adult women is influenced by menstrual changes.

6. Androgynous women and androgynous men show enhanced spatial

ability, while "feminine" women and "masculine" men show non-enhanced spatial ability. This trend was clear whether degree of "masculinity", "femininity" and androgyny was measured by self-reports or derived from physical appearance.

7. Women with Turner's syndrome are distinctly feminine and have extremely low spatial ability. Short-term treatment of such women with female sex hormone seems to restore their spatial ability to the normal female-level, while long-term treatment may lead to extremely low spatial ability.

8. Prenatal exposure to androgen-like agents may enhance masculinization and is associated with enhanced postpubertal spatial ability in women. On the other hand, androgen-insensitivity and surplus estrogen feminizes men and is associated with depressed spatial ability.

9. Men with protein-deficiency are feminized by surplus estrogen and score like normal women on spatial tasks.

10. Abnormal levels of plasma testosterone and/or abnormal steroid conversion in men with either a supernumerous Y-chromosome and a pronounced masculine phenotype or continuous experimental veneal infusion of testosterone in normal men are concomitant to depressed spatial ability, while low testosterone level in feminized men with Klinefelter's syndrome is also associated with depressed spatial ability.


## DISCUSSION

Numerous attempts have been made previously to explain the etiology and basic mechanisms for the expression of spatial ability. A variety of theoretical accounts have been proposed from the point of view of social psychology and anthropology, general psychology, psychogenetics, neuro-psychology and psycho-endocrinology, often, but not always formulated in a developmental context. An exhaustive and detailed review of all these theories is beyond the scope of this Monography. However, a brief presentation of main principles of prominent theories will be given to reflect current accounts on causal mechanisms for the expression of spatial ability, followed by a discussion of shortcomings of prevailing theories. The following discussion points out that concepts other than those in use today must be developed in order to provide an adequate explanation for recent findings on spatial ability in men and women.

### Socialization and Early Differences Theories

As socialization theories dominate the research on spatial ability, they will be dealt with in some details in this section.

Some adherents of socialization theory and especially some feminist writers

consider the size of the sex difference in spatial ability trivially small (e.g. Jacklin, 1979). However, the majority of studies testify a consistent and significant sex difference in spatial ability. Furthermore, Burstein *et al.* (1980) have argued that even a small sex difference may be the result of the use of different cognitive strategies by men and women, and that such knowledge could lead to a fertile search for reliable predictor variables. Another argument in favour of minimizing the impact of the sex difference in spatial ability is that the considerable overlap between male and female distributions renders the sex difference practically unimportant. However, individuals eminent in, say, mathematics, geometry, physics, mechanics, architecture and similar subjects so heavily dominated by men, most probably are recruited from the upper percentiles of the spatial ability distribution, which are also heavily dominated by men. It remains to be proven that the observed covariation is incidental. Hyde (1981) recently made a point for the unimportance of sex as a variable in spatial ability research. She calculated the $\omega^2$ index (Hays, 1963) to see to what extent sex accounted for the significant sex difference in field dependence and in visual-spatial ability. She found that sex accounted for less than 5% of the difference, and concluded that gender is a poor predictor not only of one's performance in spatial ability tests but also of one's performance in jobs requiring these abilities. Such sweeping conclusions are simply not called for when high level performance in spatial ability and in spatial ability related occupations are considered, as witnessed in the nearly exclusive male dominance in both these areas. There are few if any female Nobel prize winners in spatial ability related areas although a calculation by Hyde indicates an expected approximate 3:1 ratio of males to females. Thus, while the statistics recommended by Hyde perhaps have some virtue at low and medium levels of spatial ability, it certainly gives grossly misleading estimates of sex as a predictor variable in the range of greatest interest for the recruitment to spatial ability related occupations. Plomin and Foch (1981) also made the point that sex accounts for only a trivial fraction of the total inter-individual variation based on studies reviewed by Maccoby and Jacklin (1974) and on their own studies (Foch and Plomin, 1980). However, Sanders *et al.* (in press), specifically adressing the conclusion by Plomin and Foch, demonstrated that sex accounts for only 2% of the variance on the Card Rotation test, but for no less than 16% of the variance on the Mental Rotation test. Sanders *et al.* therefore argued that sex is a non-trivial variable in some areas of intellectual functioning. In passing it can be mentioned that the new theory of spatial ability to be presented later in this Monograph assigns far less importance to the average sex difference than to the tremendous inter-individual differences in spatial ability.

   Most of the relevant social learning theories aiming at explaining the average sex difference in spatial ability share two basic assumptions: One is that boys and girls are reared differently, and the other is that socialization

affects spatial ability. The theories vary, however, with regard to the emphasis they put on internal and external influences. Some social learning theories operate with *tabula rasa* models while others postulate stable early differences upon which societal forces exert an effect.

According to *tabula rasa* models (e.g. Bandura and Walters, 1963; Michel, 1966) sex differences appear only because of separate cultural norms for boys and girls. Sex-appropriate behavior is reinforced according to norms. Generalization then takes place, so that situations similar to those in which the reinforcer occurred will also promote sex-typic behavior. According to social learning theory, the child may also copy the behavior of the same-sex parent through observational learning and generalize these experiences. Three implications of such social learning theory are that only behavior that is shaped by reinforcement will appear, that behavior can be changed immediately at any time and without restrictions provided the reinforcing conditions are changed, and that children will resemble their same-sex parent more than their opposite-sex parent.

Kohlberg (1966, 1969) has presented another version of social learning theory that emphasizes the meaning of first establishing gender identity and learning what is "male" and "female", and then altering those cognitive structures that permanently change the child's way of perceiving so that sex-typed activities become differentially reinforcing. The concepts of social learning theories have been applied to the development of spatial ability (see Hoyenga and Hoyenga, 1979; Maccoby and Jacklin, 1974), but as a rule they are more concerned with personality development than with cognitive development.

Other theories based on early differences between the sexes have considered the development of spatial ability and have claimed that boys generally move more freely around in their surroundings than do girls. According to these theories locomotion furthers spatial ability. As girls are said to be subjected to more restrictions in moving around it is reasoned that sex-specific rearing hampers the development of spatial ability in females.

Piaget and Inhelder (1956) considered early sensory-motoric exploration to be essential for later establishment of "practical" and "representational" space. An implication of their theory is that early sex differences in exploration lead to sex differences in spatial ability. Witkin *et al.* (1962) considered biological as well as social factors. They put more emphasis on social factors and especially on mother–child relationships. According to Witkin, restrictive rearing of girls leads to impaired psychological differentiation and to social and perceptual "dependency" as reflected in an authoritarian attitude and in low scores on field-dependency indicators containing spatial components. Berry (see Witkin and Berry, 1975) adopted this line of reasoning in order to interpret cross-cultural data on sex differences in spatial ability.

Maccoby (1966) assumed that society confers different motivational pattern in the two sexes thus explaining sex differences in spatial ability. Passive dependency and orientation towards social cues are encouraged in girls and are difficult to give up again. A passive and dependent tendency is then assumed to make it difficult for girls to cope with tasks, requiring spatial ability which require a certain amount of assertiveness. Impressed by the observation that very aggressive boys also demonstrate low spatial ability, Maccoby assumed that it is necessary to be positioned in the middle of the passive dependent–active aggressive dimension, i.e. to be an aggressive girl or a passive boy, in order to show high spatial ability. Garai and Scheinfeld (1968) speculated that an early difference in sense modality might explain the tendency for girls to develop superior verbal ability and for boys to excel in spatial ability. This idea is elaborated upon by McGuinness (1976) and by Sherman (1974), although in quite different ways. McGuinness wondered whether the greater visual sensitivity of boys can explain their object-orientedness, and considered in addition a sex difference in arousal. Sherman also noted the earlier verbal development of girls, and suggested that this advantage can "bend-the-twig" in such a way that girls become accustomed to try to solve spatial tasks by verbal means, which would be an inferior approach. According to Sherman, the lack of linguistic ability in boys bends-the-twig towards solving spatial tasks without verbal assistance. These different approaches to solving spatial tasks by boys and girls could be reinforced by cultural stereotypes.

There are a number of serious shortcomings in social learning theories (Harris, 1979; Nash, 1979). One shortcoming relates to the social-learning dictum that boys and girls are reared in radically different ways. In a review of the literature, Maccoby and Jacklin (1974) found little evidence to support the notion that one sex receives more reinforcement than the other with regard to social learning. Their survey showed a high degree of uniformity in the socialization of the sexes. Another premise of socialization theory that deserves close attention is that girls are kept under stricter command than are boys. Maccoby and Jacklin found, however, that if a consistent difference existed at all, it was rather the boys who had the most intense socialization experiences.

Another idea of socialization theory is that girls who consider tasks with a spatial component such as mathematics for example to be a male domain, will voluntarily reduce their competence, and that this explains the sex difference in this area. It has been demonstrated, however, that interest in mathematics is not an important variable for understanding the sex difference (Kelly, 1978).

It has been suggested by socialization theory, that induction of a male identity promotes spatial skills. Aside from the lack of experimental evidence for this interesting proposal, the suggestion raises the problem of what makes for male identity: Social and/or other factors. There is ample evidence to show

that social factors alone cannot account fully for gender identity. Thus Imperato-McGinley *et al.* (1974) and Imperato-McGinley *et al.* (1976) observed that androgen exposure could change gender identity at puberty from female to male despite many years of female rearing, while Savage *et al.* (1980) found that exposure of the brain to androgens during foetal life and there-after has pronounced effects on gender identity. Of course, such observations do not indicate that social variables are without effect on gender identity (Ehrhardt, 1981), but rather that other factors should also be considered.

Sex role research also suffers from lack of objective definitions. Terms like masculinity and femininity are often treated as though they designate opposite poles of one dimension. Whalen (1974) has argued, however, that masculinity and femininity are not unitary processes, and suggested that these terms reflect many behavioral dimensions that can be independent.

Another problem with socialization research is that the critical component considered to be responsible for influencing spatial ability is seldom specified in any objective way. The independent variable is sometimes said to be "strict rearing", "parental pressure", "cultural norms", "teachers' expectation", "fear-of-failure", "girls soon learn to know", and so forth. Sometimes one variable is introduced as dependent and as independent at the same time, for example when aggression is said to be guided by societal forces, and also to affect spatial ability (Maccoby 1966). Although not improbably, the suggestions cannot be easily tested by the experimental designs used most often in socialization research. Those designs are almost exclusively based on correlations, and it is well-known that correlations do not imply causation. Nevertheless, from the common observation that boys tend to be more physically active than girls the conclusion is drawn that a high degree of locomotion facilitates spatial ability. But it could equally well be that a third unknown factor was responsible for both locomotion and spatial ability or that spatially gifted children found it more rewarding to move around more than less gifted children. Surely, various hypotheses could be generated, but not easily tested in the all too common correlational design. A further problem with prevailing socialization research is that parental power is seen almost exclusively as an independent variable, although this is not necessarily the case. Any father who has given a "boy's toy" to his daughter and has witnessed sadness in her eyes has received strong reinforcement that will surely influence what he gives her next time. Further it is questionable that children's sex-typed toy preferences are the result of conscious attempts to act in accordance with sex-role stereotypes (Eisenberg *et al.*, 1982) and that play with "boy's toy" supports spatial ability development to the extent that the sex difference in spatial ability is fully explained. Obviously, children also influence their parents, and sometimes quite effectively, so the notion that parental expectations or sanctions control children's behavior in a

one-way fashion, as much socialization research assumes, is not generally correct.

Another weakness of socialization research is the lack of specification of the mechanisms thought to mediate the socialization effect on specific cognitive abilities. The effect of all environmental experience is of necessity mediated via the central nervous system. Socialization research usually neglects this truism therefore, also ignoring the fact that genetic variation procures notable individual differences in central nervous information processing (and in personality) that may have a profound effect on how a certain environmental condition is interpreted by various individuals. Socialization studies relate for example, degree of parental pressure to cognitive ability, while considering intervening organismic variables in the pressure-ability route as "noise", and this is a questionable procedure.

In summary, it seems fair to conclude that the two basic assumptions of socialization of spatial ability are still by and large, lacking supportive evidence. The two sexes are not treated as differently as socialization theory claims. It has not been documented in any convincing way that one-way parental pressure affects spatial ability. Furthermore, socialization research usually lacks objective definition of the variables, suffers from conceptual confusion, applies weak correlational approaches that make it virtually impossible to derive causal links, and never specifies the mechanisms mediating the postulated socialization effect to the cognitive level. It is likely that as long as these serious shortcomings exist in socialization research, such studies will continue to generate more heat than light.

When shortcomings of socialization theories are taken into account, it is clear that they are unable to explain many findings obtained in research on spatial ability in men and women. For example, how can a sex difference in spatial ability be virtually absent in the prepubertal period and how can it appear around puberty? How does a sex difference in spatial ability become stabilized shortly after its first appearance and how can children tend to be more similar in spatial ability to their opposite-sex parent than to their same-sex parent? How can hormonal treatment change spatial ability in women with Turner's syndrome, how does spatial ability tend to cycle with the menstrual cycle, and how is early and late maturation connected to the expression of spatial ability?

*Genetic Theories*

Spatial ability is under some genetic influence (Vandenberg, 1967). In 1943, O'Connor advocated the theory that spatial ability is an X-linked recessive trait (XRT). The XRT theory is simple and allows for a number of precise

predictions that can be tested readily. According to the XRT theory a "good" gene for the enhancement of spatial ability exists in the gene-pool of a given population. As the gene is recessive, men need only one allele on their single X-chromosome to show enhanced spatial ability, while women need one allele of the gene on each of their X-chromosomes. The chance that a given individual in the population will receive two beneficial genes for enhanced spatial ability is the square root of receiving one gene. The XRT theory therefore predicts that fewer women than men will show enhanced spatial ability. Under random mating and a recessive gene frequency of $q = 0.5$ (the frequency that best explains the mean sex difference and the shape of the male and female distributions), 50% of males are expected to show enhanced spatial ability, while only 25% ($q^2$) of females are expected to exceed the male median. The XRT theory also predicts a certain pattern of familial cross-sex transmission of spatial ability. Thus the father-son correlation for spatial ability will be zero, because fathers do not transmit X-chromosomes to their sons. On the other hand father–daughter correlations will be higher than mother–daughter correlations, because whenever the father possesses the spatial allele, he will show enhanced spatial ability and he will necessarily transmit this gene to his daughter. Contrariwise the mother may be carrier for the spatial allele and thus not show enhanced spatial ability phenotypically, but she may nevertheless transmit to her daughter the X-chromosome with the spatial allele. Finally, while the father-son correlation will be nil, the mother-son correlation will be positive because the son must necessarily inherit his mother's X-chromosome. The mother may be carrier for the allele and may not show enhanced spatial ability although she might transmit to her son the recessive allele for enhanced spatial ability. The son then shows enhanced spatial ability.

A number of studies confirmed the indicated pattern of cross-sex correlations (Bock and Kolakowski, 1973; Corah, 1965; Hartlage, 1970; Stafford, 1961), and Yen (1975) observed patterns of same-sex sibling-correlations that partly confirmed the XRT theory. Goodenough *et al.* (1977) investigated whether brothers having the same allele at two given marker loci on the X-chromosome (Xg(a) blood-group and red-green colour blindness) were more similar on spatial ability tasks than were brothers having different markers on these loci. Out of a battery of seven cognitive tests a possible linkage was observed between Rod-and-Frame and Embedded-Figures Test performance and the Xg(a) marker.

There are serious problems with the XRT theory, however. A number of recent studies did not show the predicted correlations (Bouchard and McGee, 1977; DeFries *et al.*, 1976; Guttman, 1974; Loehlin *et al.*, 1978), and Boles (1980) showed that the significant results in the earlier studies could be due to chance variation. Bock (1967) found that fathers with enhanced spatial ability did not always have daughters with enhanced spatial ability. Furthermore, the

linkage study by Goodenough *et al.* (1977) operated with a small sample size and made numerous correlations.

Studies on women with Turner's syndrome also point to shortcomings of the XRT theory. About half of women with Turner's syndrome have karyotype 45,X (Nielsen *et al.*, 1977). According to the XRT theory spatial ability is expected to be as enhanced in such women as in normal men who also have only one X-chromosome. However, this is not the case. On the contrary, Turner's syndrome women have extremely depressed spatial ability (Nielsen *et al., ibid.*; Nyborg and Nielson, 1979, 1981*a*). Some women with Turner's syndrome lack only part of the second X-chromosome. The XRT theory predicts lower spatial ability in this group than in karyotype 45,X women, because at least a number of these women can be expected to need two alleles of the spatial gene for the enhancement of spatial ability. However, the two groups perform identically on spatial tasks (*ibid.*). Finally, Turner women who lack the second X-chromosome in only part of their body cells, or are mosaics with isochromosome X, were found to have spatial ability scores indiscriminable from those of other Turner women (*ibid.*).

The XRT theory can be tested further in two other abnormal groups, namely men with an extra X-chromosome (47,XXY, Klinefelter's syndrome) and men with an extra Y-chromosome (karyotype 47,XYY). Men with an extra X-chromosome would be expected to have lower spatial ability than normal men, and score like normal women, since the Y-chromosome is according to the XRT without effect on transmission of spatial ability. Nyborg and Nielsen (1981*b*) observed a tendency for Klinefelter men to score lower on spatial ability tasks than normal women, which seems to support the theory, but men with double Y obtained a very poor score for spatial ability, which is clearly against the XRT theory.

The recent lack of empirical support for the XRT is, of course, a serious problem. But there are also more general problems with genetic theories for spatial ability. While socialization theories are reductionistic in the sense that they restrict the range of explanatory variables to the social sphere, the XRT theory is reductionistic in that it restricts the causal variable to the genetic sphere. A full account for the enhancement of spatial ability may not necessarily be found in either sphere alone. Some genetic theories take environmental factors into account, and traditional heritability estimates usually contain, besides genetic factors, also environmental variables and further co-variation between variables. Recent path-analyses also contain refined accounts of ingoing variables and the correlations between these variables. The trouble with such analyses is, however, that the relation between variables is considered in terms of statistics. This usually implies that the relation between variables is assumed to be the same in all persons in the population studies, which is not necessarily the case. Another problem with most genetic accounts is that they typically treat interactions between

environmental and genetic variables in a purely statistical sense that fails to correspond to developmental processes going on inside the individuals in question. Furthermore, genetic theories show a tendency to leave important variables undefined. What exactly is "a good gene" for spatial ability? What have we learned about specific environmental variables when a genetic analysis shows that "environment" can account for, say, 50% of the variability? Finally, traditional genetic theories do not account for the mechanisms behind observed transmission of spatial ability. While recent advances in molecular biology may give some hope that the pathways from genes to behavior can be better understood, it seems fair to conclude for the time being, that genetic accounts such as the XRT theory have been unable to provide an adequate explanation for the experimental findings on spatial ability in men and women.

*Hemispheric Specialization Theories*

It has been speculated that the brain in females and males may differ anatomically and that such differences could explain sex differences in behavior. While anatomical differences between the brain of men and women have been found, those concerned with lateralization have been slight (e.g. McGlone, 1980; Wada *et al.*, 1975; Witelson and Pallie, 1973), and their functional significance remains unknown. Another idea that has received attention is that functional differences in brain organization in females and males may explain sex differences in spatial ability. Such neuropsychological theories of hemispheric specialization seem to have three basic assumptions; (1) that hemispheric specialization is related straightforwardly to the expression of spatial ability; (2) that the two sexes differ in degree of hemispheric specialization; and (3) that the development of a sex difference in hemispheric specialization explains sex-specific development of spatial ability. Some evidence for the first two assumptions is at hand. Damage to certain areas in the right side of the brain lowers spatial ability in both sexes, but probably more so in men. Damage to the left side of the brain is not likely to lower spatial ability in men, while it tends to do so in women (see review by Harris, 1978, 1981; McGlone, 1980). Thus spatial ability in general seems related more to right hemispheric function in men than in women, but this relationship might not necessarily show up on all spatial tasks.

The question of *when* males and females begin to differ in hemispheric specialization is more controversial. This is unfortunate, because it can, according to lateralization theory, be expected that development in cerebral functional specialization governs development of spatial ability. According to one theory (Buffery and Gray, 1972) earlier and quicker brain lateralization occurs in girls than in boys who tend to be bilaterally organized. Witelson

(1976) found, however, that boys are unilaterally organized at age 6, while girls still showed bilateral organization at 13 years of age. Further evidence indicates that boys are more lateralized at birth or that they surpass girls in unilateral organization in childhood or young adulthood (see Harris, 1978, 1981). Recently McGlone (1980) found male brains to be more asymmetrically organized than female brains for both spatial and verbal functions. Her review indicated, however, that these trends are significant only in adulthood and are seen rarely in childhood.

Lateralization theory contains some mutually exclusive ideas about when a sex difference appears. It is claimed, for example, that girls start by being more lateralized than boys on verbal tasks (Kimura, 1967) and also that boys start by being more lateralized than girls (Levy and Reid, 1978). Results from studies on a sex difference in lateralization of spatial tasks are equally confusing (Newcombe, 1981). There are those who believe that the sexes do not differ in lateralization before adulthood, while Waber (1977a) observed that lateral specialization is higher in late maturers, as is spatial ability, than in early maturers, regardless of their sex. Mayes (1982), however, was unable to confirm the hypothesis that both cultural and sex differences in spatial ability relate to a particular pattern of hemispheric specialization. Clearly, the question of when and how a sex difference in lateralization takes place needs further attention. Also the question of what to measure requires consideration. In the spirit of Gur and Gur (1980), O'Connor and Shaw (1978) measured *hemispheric activation* (EEG coherence values for the alpha frequency band) rather than *lateralization of cognitive capacity* and related this measure to spatial performance in the Rod-and-Frame Task. Witkin and Asch's (1948) confounded measure for field dependence showed no relation to the alpha frequency measure for hemispheric activation. However, taking advantage of Nyborg's (1974, 1977) analysis of the spatial constituents of the Rod-and-Frame Test, O'Connor and Shaw were able to demonstrate a specific association between hemispheric activation and frame dependence.

Researchers in the area of hemispheric specialization surely have to solve a number of problems before clear results can be expected to emerge (Hiscock and Kinsbourne, 1977). Bryden (1979) notes that a left-right difference in tests for laterality does not necessarily indicate a left-right difference in cerebral organization. Vandenberg and Kuse (1979) suggest that both hemispheres are probably required for optimal performance in spatial as well as in verbal tasks. Problems with validity and reliability of instruments for measuring hemispheric specialization, and with the experimental designs forced Bryden (1979) to note that the proper developmental study has not yet been done, that most studies can be criticized for poor procedures, lack of replicability, or too small samples, and that test results can be radically changed by a variety of sex-related individual differences in the deployment of attention, problem-solving "set", and cognitive strategy. The pessimistic conclusion of Bryden's

review was that either there are no meaningful sex-related differences in cerebral asymmetry at all, or that the differences emerge only after puberty. The last idea seems at least consistent with the notion that the development of hemispheric specialization relates to spatial ability, since sex differences in spatial ability typically appear around puberty. However, Kinsbourne and Hiscock (1977) consider it unlikely that lateralization develops with age.

## Multifactorial Model

Spatial ability is a complex trait to which numerous variables may contribute. Recognition of the complexity of spatial ability, perhaps in combination with acknowledging the shortcomings of most other theories, led DeFries *et al.*, (1976) to suggest a multi-factorial, sex-modified threshold model to explain sex differences in spatial ability. According to their model, numerous socialization, genetic, neural, and hormonal factors might combine additively to enhance spatial ability. DeFries *et al.* explained the sex difference in terms of men having a lower threshold, i.e. needing fewer factors in order to show enhanced spatial ability. While a serious problem with some of the theories discussed above is that they are too restrictive in their choice of variables, the multi-factorial, sex-modified threshold model may be too permissive in that it allows too many variables to influence spatial ability. To say that many factors may influence the phenomenon under study is only to acknowledge a problem, not to explain the phenomenon. To say that men have a lower threshold for showing higher spatial ability than do women, raises the question of why?

## Present Hormonal Theories

A number of observations indicate that at least certain aspects of cognition might also be under hormonal influence. Broverman *et al.* (1968) formulated a theory for steroid-cognition relations. Their theory considers both estrogen and testosterone to influence adrenergic functioning in the brain by regulating the monoamine oxidase (MAO) enzyme level. According to their theory, changes in MAO cause changes in spatial ability. Both estrogen and testosterone inhibit MAO, but estrogen is assumed to be a stronger inhibitor than testosterone. Since women have more estrogen than men, their MAO is more inhibited, making women more adrenergic-dominant, stronger automatizers, and thus lower in spatial ability. The weak inhibitor, testosterone, makes men more cholinergic-dominant, weaker automatizers, and thus higher in spatial ability.

The hormonal model has several weaknesses. Singer and Montgomery (1969) found the postulated relation between cognitive differences and neuro-

transmitter ratios to be based on a number of assumptions which either are contradictory to facts or simply unsupported by empirical evidence. Also the dichotomy of an "adrenergic-activating" sympathetic nervous system and a "cholinergic-inhibiting" parasympathetic nervous system was found by Singer and Montgomery to be incorrect. They stressed that it is no longer adequate to propose biochemical models of CNS functioning without some attempt to specify a locus for the proposed phenomena. Parlee (1972) noted that the neural-cognition relationship proposed by the Broverman group is not adequately demonstrated, and that the literature-based part of the model is too selectively represented. Harris (1978) regretted that most of the evidence directly supporting the theory is from animal studies.

   Other explanations have been offered to account for observations on relations between hormones and cognition. Bock and Kolakowski (1973) were concerned about the inability of the XRT theory to explain a number of observations, and speculated as to whether testosterone production may be involved in some way in the expression of spatial ability, and Hughes (1980) also speculated on the effect of testosterone on spatial ability. Hier and Crowley (1982) actually did a study on the effect of androgen deficiency on spatial ability. Nineteen men with idiopathic hypogonadotropic hypogonadism were scored on Block Design, Embedded Figures, and the Space Relations subtest, and their results were compared to those of men with acquired hypogonadotropic hypogonadism and of normal men. Men with idiopathic hypogonadotropic hypogonadism did significantly less well on the spatial tasks than the controls, but their testicular size correlated positively with performance on both Block Design and Embedded Figures Test on the 5% level. Hier and Crowley took these results to mean that androgenization by testosterone or one of its metabolites is essential for the enhancement of spatial ability, and further that the effect is exerted at or before puberty, because later androgen-replacement therapy could not compensate for the earlier deficiency, and because men with the acquired form of hypogonadotropic hypogonadism demonstrated intact spatial ability. Englander-Golden et al. (1976) wondered whether menstrual changes in hormones, influence cognition. McGee (1979) suggested that there might be an optimal estrogen-testosterone balance for high spatial ability, an idea that is also alluded to by Petersen (1976) who proposed that spatial ability is curvilinearly related to the testosterone/estrogen ratio such that intermediate levels benefit spatial ability. Waber (1976, 1979) suggested that sex differences in spatial ability might be explained by a hormonal inhibition or cerebral re-structuralization around puberty.

   In summary, the notion of a connection between sex hormone levels and spatial ability has been in focus recently. In the following, this notion will be considered further in an effort to account for aspects of spatial ability in terms of individual variations in sex hormones.

# NEW THEORY

## *Introduction*

It is clear that the conclusion to be drawn on the basis of the discussion of available theories for explaining the development of spatial ability is that none of them is fully satisfactory. The theories suffer in varying degree from terminological and conceptual difficulties, inconsistent results, lack of experimentally well-controlled designs, small samples, unreliable and/or invalid instruments. Many of the hypotheses proposed are certainly plausible, but as formulated they are often impossible to test rigoristically. Mechanisms responsible for mediating the postulated effects are typically left unaccounted for. Some of the theories neglect individual variation in that they imply that all boys, all girls, all right cerebral specialized, all early maturers, or all children reinforced this or that way, will necessarily show a certain predictable degree of spatial ability. Some theories center on causal agents within a narrow frame. Thus socialization theories naturally advocate social factors, but generally neglect biological factors. On the other hand genetic theories often treat environmental variables only in terms of statistical interactions. Most theories are not able to account for the timetable for the development of spatial ability. As a result, they fail to provide a satisfactory explanation for the appearance of sex differences in spatial ability around puberty, even when considering the female lead in somatic pubertal development, and how the male lead in spatial ability appears around puberty and becomes stabilized shortly afterwards. Most theories cannot explain the occurrence of alterations in spatial performance with the menstrual cycle or how spatial ability becomes depressed in some and enhanced in other groups with sex hormone abnormalities. Thus, new theories are needed in order to provide an adequate explanation for the findings on spatial ability in men and women.

New theories must fulfil certain criteria in order to be an improvement compared to existing theories on spatial ability in men and women. They must be of an integrative kind. They must be more specific than available multifactorial theories. They must take possible relations between hormone levels, physical development, cerebral maturation and hemispheric specialization into account. They must acknowledge the temporal relation between physical maturation rate and spatial ability. They must explain the level of spatial ability seen in groups with hormone abnormalities. They must provide an explanation for monthly changes in spatial ability in menstruating women, for the general superiority of spatial ability in adult men compared to adult women, the high spatial ability of "masculine-looking" women and "feminine-looking" men, as well as the low spatial ability of "feminine" women and "masculine" men. In addition, new theories must use well-defined

and accurately quantifiable causal variables that can be delineated in detail by specifying their values, locus of action, mechanism through which they exert their effects, and their impact on the dependent variable, namely the expression of spatial ability. Furthermore, the theories should be formulated in such a prospective manner, that allows for the generation of a number of hypotheses that can be subjected to rigoristic experimental testing. On the other hand, the theories should be conceived in such a way that they forbid the outcome of alternative sets of hypotheses (Popper, 1973). Finally, the theories should be able to encompass environmental and biological variables in a frame of reference that goes beyond statistical interaction. Clearly, much essential knowledge about the etiology of spatial ability is presently lacking, but it may nevertheless be possible to develop a better theory than those presently available.

## Developmental and Functional Relations of Plasma Steroids

The results of the studies reviewed suggest that estrogen and testosterone may play a role for the expression of spatial ability. It is therefore of interest to determine the plasma values of these two steroids in men and women and further to delineate their functional relations in order to arrive at a new theory of spatial ability in men and women.

Estradiol ($E_2$) is the biologically most active estrogen. Plasma $E_2$ values are similar in the two sexes up to 10 years of age, and are no higher than 10 pg/ml during that period (Marcus and Korenman, 1976). Eleven year old boys (Tanner's stage 1: Tanner, 1962) have a plasma $E_2$ value of $4.8 \pm 1.5$ pg/ml, while girls at the same age have $E_2$ values of $9.8 \pm 2.4$ pg/ml (Aususingha et al. 1974; Jenner et al., 1972). This sex difference in plasma $E_2$ increases rapidly between 11 and 13 years of age. Thus 13 year old boys typically have plasma $E_2$ values about $21 \pm 4.9$ pg/ml, while 13 year old girls have a plasma $E_2$ value of $65.7 \pm 35.3$ pg/ml (see also Jenner et al., 1972). $E_2$ values between 20 and 35 pg/ml are typical for adult men (Hawkins and Oakey, 1974; Marcus and Korenman, 1976) and the plasma $E_2$ level varies with the menstrual cycle in adult women around a mean value of $40 \pm 3$ pg/ml (Vermeulen, 1976). It is noteworthy, however, that the plasma $E_2$ value is relatively low (20–50 pg/ml) during the early phase of the menstrual cycle, while it is relatively high (50-100 pg/ml) during the later phase of the menstrual cycle (Abraham et al., 1971; Klaiber et al., 1974). The plasma $E_2$ values drop markedly at menopause to about $20 \pm 1$ pg/ml, which is very similar to the value for ovariectomized women (Vermeulen, 1976). After menopause, the plasma $E_2$ level in women falls to a value slightly lower than that in men of same age (Hawkins and Oakey, 1974).

There are prenatal and perinatal sex differences in total plasma testosterone

(T). T in amniotic fluid is relatively high in male fetuses from week 15 to 26 with peak values at weeks 17, 22, and 24 of gestation, and also 4–6 hr (see Stahl *et al.*, 1978), and 2–6 months after birth (Sizonenko, 1978). During infancy and childhood there are only small sex differences in plasma T up to about 12 years of age (Ducharme *et al.*, 1975). In the period from 12 to 17 years a ten- to twenty-fold increase in T occurs in boys with the largest increase between 12–14 years, while the T value of girls only increases 2- to 3-fold during that period. The plasma T level in girls from 0 to 6 year old is about 15 ng/100 ml. It increases to 25 ng/100 ml at 8 years, and to 65 ng/100 ml at 17–25 years. For boys, however, while plasma T is below 40 ng/100 ml before 10 years of age, it raises sharply to between 550–650 ng/100 ml in adulthood (Doering *et al.*, 1975; Stahl *et al.*, 1976; Wieland *et al.*, 1980). T levels fluctuate only slightly during the menstrual period in women. Abraham (1974) observed a plasma T value of 20–30 ng/100 ml in the early folicular phase, and an increase to 50 ng/100 ml at midcycle, and a decrease to 30 ng/100 ml in the later menstrual phase followed by a new peak. Plasma T level is 10 times lower in castrated men than in normal men, while the plasma T level in castrated men is close to that seen in adult women (Bennett, 1976). After menopause, plasma T levels in women become stabilized around 30 ng/100 ml, while lower plasma T levels (approx. 12 ng/100 ml) are usually present in ovariectomized women (Vermeulen, 1976).

In adult women over 90% of circulating $E_2$ arises from ovarian secretion (Baird *et al.*, 1968) while in men 50–75% of circulating $E_2$ arises from peripheral conversion of T and other androgens (Calabresi *et al.*, 1976; Gupta *et al.*, 1975; Longcope *et al.*, 1969; Marcus and Korenman, 1976). Consequently, plasma $E_2$ in men is determined largely by mechanisms responsible for androgen secretion (see, however, Johnson *et al.*, 1971). Furthermore, Longcope *et al.*, (1969) observed that peripheral conversion of androgens to estrogens is greater in men than in women. Adiposity and age also influence the peripheral conversion rate. Some of the peripheral loci for conversion have been determined, but more studies are needed in order to fully understand the control mechanisms involved (Marcus and Korenman, 1976).

A dynamic antagonism between $E_2$ and T has been found in which the presence of the one may inhibit the effect of the other. Most details on the biochemistry of this reciprocal inhibition are not known, but it has been suggested that testosterone-estrogen binding globulin may be involved in the process (Marcus and Korenman, 1976). Only the free part of the total plasma steroids is biologically active at the target organs which makes their plasma binding affinity of interest. August *et al.*, (1969) noted that the testosterone binding affinity values were higher for prepubertal boys and girls than for adults, and that the values were higher for women than for men both before and after puberty. Finally, numerous animal and human studies show that

early $E_2$ or T priming may radically and permanently change later steroid metabolism and/or production. $E_2$ treatment may for example reduce testicular size and hampers steroid output.

In summary, the peripheral $E_2$ value in adult women is largely determined by the monthly cycling ovarian production. In contrast, the plasma $E_2$ value in adult men is influenced markedly by peripheral conversion of T to $E_2$. The conversion rate is affected also by adiposity and age. The biologically active fractions of total plasma $E_2$ and T are determined by reciprocal inhibition and by inhibition or facilitation by other substances in both sexes. The plasma levels of $E_2$ in the sexes begin to differ at 11 years of age, and the difference increases during the following 2–3 years so that adult women have plasma $E_2$ values between 20–100 pg/ml while men range between 20–35 pg/ml. However, during the early menstrual phase, plasma $E_2$ levels in some women approach the lower average plasma $E_2$ level seen in most men. The menopausal inhibition of ovarian steroid output brings the female plasma $E_2$ level down to, or even below, the male average. T differentiates the sexes hormonally already in the pre- and perinatal period by relatively distinct surges probably of testicular origin. Then after childhood, plasma T levels rise to about 600 ng/100 ml in men and to about 40–60 ng/100 ml in adult women.

## Central Loci of Hormonal Target Organs

One way to determine the central loci of hormonal action is to map the location and degree to which steroids accumulate in the central nervous system (CNS). Various techniques are at hand to do this. A common procedure is to treat animals with controlled doses of steroids and then to disect the brain in order to look for systematic variation of CNS steroid concentration. In addition, radioactive labelling of injected steroids can be used for tracing the pattern of uptake. These studies show that certain "primitive" or "old" parts of the brain are especially prone to accumulate circulating steroids. The preoptic-hypothalamic area and the limbic system concentrates $E_2$ at about 10 times the plasma value (Dörner and Kawakami, 1978; Eisenfeld, 1970; Eisenfeld and Axelrod, 1966; Endröczi, 1978; Flores et al., 1973; Kato and Vilee, 1967; McEwen, 1976; McEwen and Pfaff, 1970; McGuire and Lisk, 1969; Pfaff, 1968; Stumpf and Sar, 1978; Zigmond and McEwen, 1970). Accumulation of $E_2$ in the CNS takes place within hours, and appears to occur via similar $E_2$ receptors in male and female brains (Maurer, 1978; McEwen, 1976; Whalen, 1974; Zigmond and McEwen, 1970).

$E_2$ may also accumulate in the CNS from central conversion of T (Flores et al., 1973; Kato et al., 1970; Knapstein et al., 1968), and effects of T on the cell nucleus appear to be mediated primarily by central conversion to $E_2$ (Goto and Fishman, 1977; Stumpf, 1970). Plasma $E_2$ and $E_2$ converted from T most

probably have different distributions in the body as the enzymes necessary for the conversion are not found everywhere. T is converted in the preoptic areas, the hypothalamus, and the amygdala but not in the pituitary gland (McEwen, 1976). Converted $E_2$ is likely to be biologically active in the CNS because it is not subjected to systemic dilution and metabolic degradation before reaching sites of action (Flores *et al.*, 1973).

Of course, the pattern of accumulation of $E_2$ and T in the CNS does not necessarily reflect the central sites of biological activity (Endröczi, 1978). Nevertheless, the findings on the central distribution and conversion of sex hormones suggest that "primitive" brain areas are involved in their actions. The evidence also indicates that one pattern of influences is based on plasma $E_2$, while another pattern is based on T converted to $E_2$. Since plasma T level is many times higher in men than in women, it seems likely that conversion of T to $E_2$ at the level of the CNS — other things being equal — has more of a central effect in men than in women and that perhaps this effect differs from the one based on uptake of plasma $E_2$.

## *Central Mediating Mechanisms*

At least three hypotheses have been proposed to account for steroid effects on the CNS: (1) The responsivity hypothesis; (2) the organization hypothesis; and (3) the activation hypothesis.

### *The responsivity hypothesis*

Beach (1945) claimed that sex hormones are best regarded not as stimuli or as organizing agents, but as chemical sensitizers which alter the stimulability of critical mechanisms within the CNS (p. 400). According to Beach, early hormonal priming will alter the responsivity of the CNS to circulating hormones in later life. Support for the responsivity hypothesis comes from studies showing that prenatal and/or perinatal exposure to $E_2$ as well as to T can modify later response to sex hormones (Balazs *et al.*, 1975; Beatty and Beatty, 1970; Ciaccio and Lisk, 1971; Edwards, 1969; Flerkó *et al.*, 1973; McGuire and Lisk, 1969). Although there are many unresolved issues on effects of early exposure to sex hormones, it seems reasonable to consider early steroid priming as of some importance for understanding later CNS responsivity.

### *The organization hypothesis*

The question of whether male and female brains differ in organization at birth or even earlier is unsettled. A "neutral-at-birth" point of view has been advocated by some (Money and Ehrhardt, 1972), while others consider

exposure to sex hormones *in utero* to organize patterns of neuronal functioning in a sex-specific way (Brawer and Naftolin, 1979; Harris, 1964; Torand-Allerand, 1976; Young, 1967). Those who postulate *in utero* effects on brain development consider sex hormones to act in the cell nucleus by triggering changes in gene expression. Alterations in gene expression induced by sex hormones will, in turn, be expected to influence protein production, neural growth and cerebral organization (Arai and Matsumoto, 1978; Cavallotti and Bisanti, 1972).

It is of interest to speculate on whether these principles can give hints to a better understanding of the processing of spatial information in men and women. Perhaps $E_2$ acts on specific aspects of brain organization rather than exerting a general unspecific effect in the brain (Goy and McEwen, 1980; Timiras, 1971). Foregoing considerations suggest that $E_2$ may influence sensory processing mainly by effects in so-called primitive brain regions.

The organizational effect of $E_2$ in the brain appears to be dose-dependent. The principle of the dose-effect relation for $E_2$ seems to be that either too much or too little of the hormone is damaging, while intermediate levels of $E_2$ seems to optimize neural development. The "too much–too little" principle may be of importance for understanding how sex hormones affect the brain (Arai and Matsumoto, 1978; Brawer and Naftolin, 1979; Döhler and Gorski, 1981; Döhler and Hancke, 1978; Goy and McEwen, 1980; Ohno *et al.*, 1974; Timiras, 1971; Torand-Allerand, 1976). Recently, Nyborg and Nielsen (1981*a*) suggested that the low spatial ability and the moderately abnormal EEG in anovulatory women with Turner's syndrome could be explained by inadequate neural development due to unphysiological low $E_2$ levels early in life. These authors then demonstrated that short-term cyclic estrogen/gestagen treatment may restore spatial ability in Turner's women to a normal female level, while long-term cyclic estrogen/gestagen therapy was associated with low spatial ability. It was speculated that short-term treatment at first speeded up the delayed neural development to a near-normal level, but that prolongation of treatment had a destructive effect on neural structures underlying spatial ability. The "too much–too little" principle may be worth keeping in mind when studying neural development and spatial ability in hormonally abnormal groups other than women with Turner's syndrome, and in normals.

Effects of early steroid priming of later target organ responsivity, and of steroid impact on neural development, could account for actions of $E_2$ on neural structures underlying spatial ability. It is assumed by the new theory that early developmental effects of $E_2$ on the originally neutral brain "set" a person-specific level of spatial competence. However, while this principle may turn out to be a necessary condition, it surely is not sufficient for understanding later expression of spatial competence, in that a number of findings such as the monthly change in spatial ability in menstruating women

indicate that temporary changes in plasma steroid level relate to transient fluctuation of spatial competence. Therefore, an acute activation hypothesis is also incorporated in the new theory in order to explain temporary fluctuations around the person-specific level of spatial competence.

*The activation hypothesis*

The new theory considers regulatory actions of sex hormones on genes to be responsible for activational effects of the hormones on spatial ability. Sex hormones may turn certain genes on or off (Jacob and Monod, 1961). Edelman (1975) suggested the following steps in steroid actions on genetic expression: (a) Penetration into the target cell; (b) steroid-specific binding to high affinity receptors; (c) temperature-sensitive activation of the steroid-receptor complex; (d) attachment of the active complex to chromatin; (e) induction of RNA and protein synthesis; and (f) the physiological expression of the induced protein.

According to Hoyenga and Hoyenga (1979) there are two kinds of activational effects; specific and generalized. The specific effect consists in hormones changing temporarily the cellular activity in a specific area of the brain, thereby making one particular kind of response to a specific environmental stimulus more likely to occur. The non-specific activational effect of hormones consists in affecting temporarily the general level of activity in rather large parts of the brain by influencing transmitter substance concentration (Kobayashi *et al.*, 1966) and/or by inhibiting an inhibitor of brain activity thereby actually facilitating general brain excitability (Broverman *et al.*, 1968) and/or by affecting the electrical activity of the brain (Gorski, 1976). T and $E_2$ seem in general to increase brain activity (Hoyenga and Hoyenga, 1979). While too little brain activity may cause poor performance on tasks requiring spatial ability, too much brain activity may also be detrimental because most spatial ability tasks seem to call for some inhibition of responding (Broverman *et al.*, 1968). The activational effect of sex hormones may take place within hours, a time span in which marked variations in blood and brain $E_2$ concentrations can occur (Zigmond and McEwen, 1970).

Responsivity, organizational and activational effects of $E_2$ and T are probably not independent events. Early steroid presetting of target organ responsivity may for example be understood at least in part in terms of an organizational effect (McEwen, 1976). Likewise, early disappearance of receptor cells due to organizational effects probably lowers later over-all target tissue responsivity. Thus, interactions between effects of $E_2$ on neuronal processes suggest that many aspects of behavior may be influenced by steroid priming, early organizational, and later activational mechanisms (see, for example, Hoyenga and Hoyenga, 1979).

With regard to the expression of spatial ability there are a number of logical possibilities for establishing a hormone-dependent time-table. One is that early steroid priming and/or neural organizing effects may show up mainly by themselves at puberty, i.e. without the assistance of later activational effects. Another possibility is that activational effects around puberty explain adult expression of spatial ability. A third possibility is that early influences by hormones might organize the brain in a way that shows up prepubertally as an inductive effect which upon facilitation will show pubertal activational effects. A number of the studies reviewed point to the last possibility. The new theory assumes that early effects of sex hormones can be favorable or detrimental for the development of those nervous tissues subserving spatial processing. These early inductive steroid effects on nervous tissues are not specific to gender but set a prepubertal level of spatial competence in accordance with that individual's cerebral $E_2$ level. At puberty and afterwards, that level of spatial ability can be either depressed permanently by the activational effects of either a continuous, too high, or too low level of steroids, or enhanced permanently at puberty by the central activational effect of a continuous adult range of optimal sex hormone levels. The new theory also postulates that the adult individual level of spatial ability may fluctuate due to modulation by temporary activational effects of short-term changes in $E_2$ levels transcending the optimal range.

*Main Characteristics of the New "Optimal Estrogen Range" (OER) Theory*

The main characteristics of the OER theory can now be summarized as follows: (1) $E_2$ is the biological agent mediating spatial ability; (2) cerebral $E_2$ is provided either directly from plasma $E_2$ or indirectly by conversion of plasma T; (3) the central locus of $E_2$ action is primarily old parts of the brain; (4) the effect of $E_2$ on spatial ability is mediated through early adjustment of later CNS steroid responsivity, and/or through early growth and organization of brain tissues essential for the processing of spatial information; (5) there is an optimal range of cerebral $E_2$ values for the maximal expression of spatial ability; (6) at puberty and perhaps also later in life activational effects of $E_2$ may lead to permanent or temporary inhibition or facilitation of spatial ability; and (7) either too high or too low cerebral $E_2$ levels minimize the expression of spatial ability.

*Application of the OER Theory to Spatial Ability of Women*

The OER theory accounts for a prepubertal rise in spatial ability in a girl, in terms of the impact of $E_2$, which imposes upon the general development.

According to the theory, $E_2$ determines each individual's level of spatial ability by exerting organizational effects on certain brain tissues. At puberty, the surge in $E_2$ production causes the concentration of $E_2$ in plasma to increase further, which leads to a reduction in spatial ability due to overactivational effects. According to the OER theory, the adult stabilization of plasma $E_2$ is responsible for each woman's general adult expression of spatial ability. The OER theory postulates also that a moderate surge in plasma $E_2$ keeps the central $E_2$ level moderate, exerts a slightly stimulating effect on relevant brain tissues and will not inhibit the expression of spatial ability to any large extent, while a large surge in plasma $E_2$ will exert a highly stimulating effect on relevant brain tissues, and thereby inhibit spatial ability markedly. By extension the OER theory explains the relatively low spatial ability of women compared to men by assuming that more women than men suffer a decline in spatial ability around puberty due to a surge in plasma $E_2$. The higher prepubertal spatial ability of early maturing girls can be accounted for by the OER theory by assuming that a higher than normal plasma $E_2$ level during that period accelerates not only body maturation (Bisanti and Cavallotti, 1972; Gupta *et al.*, 1974; Heald and Hung, 1970; Jenner *et al.*, 1972; Money and Clopper, 1974; Ramirez and Sawyer, 1965, 1966; Rosenfield, 1971; Smith and Davidson, 1968; Tanner, 1969; Warne *et al.*, 1979) but also the organization of those brain structures important for processing the spatial information, thereby bringing spatial ability to its person-specific optimal expression before puberty.

The postpubertal decline in spatial ability in early maturing girls can according to the OER theory be explained by a continuously high $E_2$ level exceeding the optimal range of central values and resulting in an activation-induced inhibition of spatial ability.

Low spatial ability before puberty and high spatial ability after puberty in late maturing girls is explained by the OER theory in the following way. First, a lower than normal plasma $E_2$ level in late maturers (Bisanti and Cavallotti, 1972; Money and Clopper, 1974) delays sexual maturation and tempers the expression of spatial ability by means of a weak organizational effect on relevant brain tissues. Then, a slow and moderate increase in plasma $E_2$ eventually results in delayed puberty preceded by a long maturational period during which extensive organizational processes take place. After puberty the relatively low adult plasma $E_2$ level ensures that the central $E_2$ level is kept within the optimal activational range for the expression of high spatial ability.

Clearly, variables other than $E_2$ levels may also influence maturational rate, so detailed multivariable studies on relations between plasma $E_2$ values, rate of maturation and spatial ability are needed to test the OER theory.

The OER theory accounts for changes in spatial performance during the menstrual cycle as follows. It will be recalled that spatial ability is inversely related to plasma $E_2$ levels during the menstrual cycle (see Review). The OER

theory considers the low concentration of $E_2$ during one phase of menstruation to lead to central $E_2$ levels near to or within the optimal range for enhancement of spatial ability for some women, while higher $E_2$ concentrations during another phase of the menstrual cycle may result in increased central excitation and impaired spatial ability.

It is of interest at this point to consider whether the optimal $E_2$ range can be estimated from available information on spatial ability of women. The decline in spatial ability of women starts around the age of 14, at which time the average plasma $E_2$ level is between 40–60 pg/ml. Adult female spatial ability is highest during the early folicular phase of the menstrual cycle at which time the plasma $E_2$ concentration is between 20–50 pg/ml, while spatial ability is lowest when the plasma $E_2$ concentration is between 50–100 pg/ml. Adult women have an average plasma $E_2$ value of 40 pg/ml and their spatial ability is slightly depressed relative to that of adult men. Taken together, these observations suggest that the optimal range of $E_2$ in plasma is somewhere between 25–35 pg/ml for the enhancement of spatial ability. However, this range is to be taken only as an estimate, and there are most probably large individual variations around these values. Factors such as early steroid priming, age-related changes in target tissue responsivity, $E_2$ production rate and metabolic clearance may all be expected to relate to the optimal $E_2$ range. Consequently, the notion of an optimal $E_2$ range of 25–35 pg/ml for enhancement of spatial ability in women must be considered only tentative.

The OER theory accounts for the effects of $E_2$ treatment on spatial ability of girls with Turner's syndrome as follows: The plasma $E_2$ concentration and the spatial ability of these girls are abnormally low. Short-term treatment with $E_2$ exerts organizational effects on their delayed brain development and this restores their spatial ability to a normal female level. Long-term $E_2$ treatment may result in a transgression of the optimal $E_2$ range and lead to an overactivation of relevant brain tissues thereby causing an impairment of spatial ability. The possibility that prolonged $E_2$ treatment may result in a neurotoxic reaction in addition to disorganization of brain tissues relevant for processing spatial information also exists (for discussion, see Nyborg and Nielsen, 1981a).

High spatial ability in girls exposed prenatally to high doses of T-like agents can be explained in several ways by the OER theory. One possibility is that the high T dose is converted centrally to $E_2$ and thereby affects CNS organization in a way that might differ from a more direct plasma $E_2$ impact. Another possibility is that T may compete successfully with $E_2$ at central receptors and render them less responsive at puberty to a surge in $E_2$. According to the OER theory, these possibilities could have a combined effect, but further speculations on this matter await studies designed specifically to test these aspects of the theory.

## Application of the OER Theory to Spatial Ability in Men

The OER theory views the relatively high spatial ability of adult men as evidence that their central $E_2$ value is slightly below or in the lower part of the optimal range. Compared to women, men have a much higher plasma T concentration. The T level in men is taken into account by the OER theory as shown in Fig. 1.
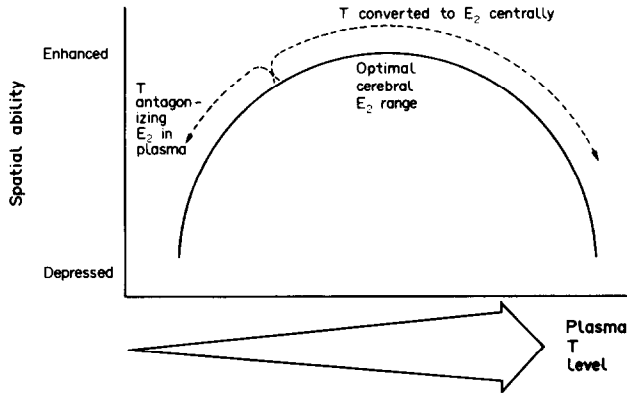


FIG. 1. Graphical illustration of possible modulatory effects of testosterone on the relation between the plasma estrogen level and spatial ability of men.

According to the theory, the lack of notable impairment of spatial ability in boys at puberty is due to a peripheral balance between $E_2$ and T. A rise in $E_2$ and in T in boys during puberty is assumed to leave their spatial ability unaffected at a level slightly below the optimal range, because of antagonistic effects of T on $E_2$. It may also be that pre- and perinatal surges of T prime male pubertal responsivity to $E_2$, or that when converted at certain brain sites T acts as a prohormone and organizes the CNS in a male direction by disposing for lateralization of spatial processes. This organizational effect of T may become functional by pubertal activation. Alternatively, antagonistic peripheral effects and central reinforcing activational effects of T on $E_2$ may balance out and leave spatial ability largely intact at puberty. Present day knowledge is insufficient to choose between these possibilities.

The OER theory accounts for relations between bodily features and spatial ability on the basis of relations between sex hormone levels and the development of secondary sex characteristics. A relative abundance of $E_2$ favors feminine features (Marshall and Tanner, 1969), while a relative abundance of T leads to masculine features (Marshall and Tanner, 1970).

Thus the high spatial ability of men displaying some feminine features in addition to masculine features can be accounted for by assuming that such androgynous men have slightly higher than normal levels of plasma $E_2$. As most men are considered by the OER theory to be situated just below the optimal range of central $E_2$ values, the slightly increased $E_2$ level in androgynous men would bring them within the optimal central $E_2$ range for the enhancement of spatial ability. Similarly, the low spatial ability of men with strong masculine features can be explained in terms of low levels of plasma $E_2$ and/or high levels of plasma T. Further studies on relations between sex hormone levels and bodily features in men are needed to test the OER theory directly.

The OER theory explains the shortlived high spatial ability of early-maturing boys in terms of their relatively high sex hormone production. Early-maturing boys may reach the central level of $E_2$ optimal for the expression of spatial ability sooner than other boys, but pass it, perhaps due to excessive central conversion of T to $E_2$. Late maturers may arrive later at the optimal central $E_2$ level, allowing more time for organizational processes to take place, but they may not be prone to surpass the optimal central range of $E_2$ values. The OER theory considers the impairment of spatial ability of clearly feminized men with Klinefelter's syndrome to be due to the relative abundance of $E_2$ present in these men (Froeland et al., 1973; Raboch and Mellan, 1978). According to the theory, the relative abundance of $E_2$, not antagonized by T due to its low level in these men, may impair the spatial ability of Klinefelter males as in normal women. In principle the same explanation applies to the low spatial ability of androgen-insensitive individuals with karyotype 46,XY. Men with the Kwashiorkor syndrome have an abundance of $E_2$ in their blood (Dawson, 1966, 1967, 1972), and according to the OER theory, their elevated plasma $E_2$ level may play a role in their reduced spatial ability compared to normal men. The hormonal picture is less clear in men with karyotype 47,XYY. While some studies of these men have shown a relative abundance of T in their blood (e.g. Ismail et al., 1968; Rudd et al., 1968; Wakeling et al., 1973), other studies have shown normal or even low plasma T level (e.g. Pitcher et al., 1974; Polani, 1972). Perhaps abnormal steroid metabolization takes place in men with an extra Y chromosome. It is accordingly uncertain, whether their low spatial ability (Nyborg and Nielsen, 1981b) can be attributed to too high or too low central $E_2$ levels. Men with idiopathic hypogonadotropic hypogonadism masculinizes normally (or nearly normally) *in utero* and are reared and educated as normal boys until puberty, when pubescence fails to occur probably due to pubertal androgen deficiency (Hier and Crowley, 1982). According to the OER theory their depressed spatial ability can be explained by a peripheral $E_2$/T imbalance bringing these men outside the optimal cerebral $E_2$ range for enhanced spatial ability.

## General Discussion

The OER theory was designed to provide an explanation for findings from experiments on spatial ability of women and men. Figure 2 presents a resume of the theory. The placement of some of the groups considered in the present
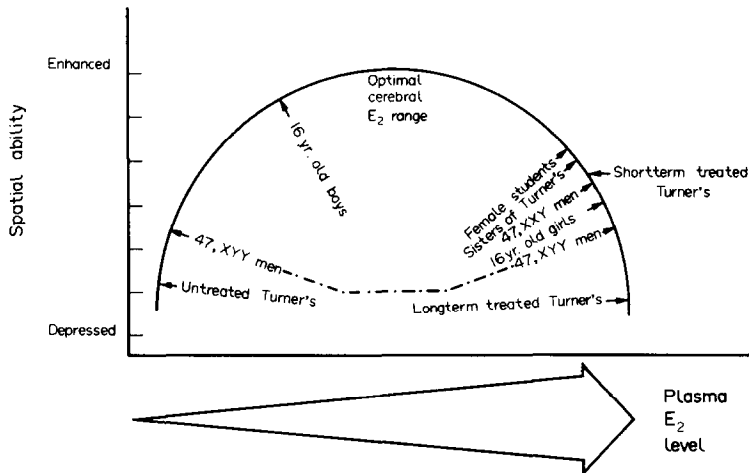


FIG. 2. Resume of the "Optimal Estrogen Range" theory showing the position of groups on the curve for the relation between the plasma estrogen level and spatial ability.

review is shown on the curve for the relation suggested by the OER theory between the plasma $E_2$ level and spatial ability as measured by the Rod-and-Frame test. Untreated women with Turner's syndrome have an abnormally low level of $E_2$ and extremely depressed spatial ability (Nielsen et al., 1977). Sixteen-year old boys are located slightly below the optimal $E_2$ range for spatial ability (Nyborg, note 1). Normal women, women with Turner's syndrome given short-term $E_2$ treatment (Nyborg and Nielsen, 1981a), men with Klinefelter's syndrome (Nyborg and Nielsen, 1981b), and long-term treated Turner women (Nyborg and Nielsen, 1981a) are located far above the optimal $E_2$ range. It is uncertain whether men with karyotype 47,XYY and depressed spatial ability (Nyborg and Nielsen, 1981b) should be placed far below or far above the optimal $E_2$ range.

The OER theory can account readily for two previously inadequately explained observations on spatial ability. One is the facilitating effect of intravenous infusion with moderate doses of T, along with inhibitory effects of high doses of T. This finding can be explained by the theory in terms of

activational effects of T. The second observation relates to the notion that spatial ability is typically defined by prevailing social learning theories as a "masculine" trait, even though spatial ability is usually higher in androgyne men than in "masculine" men. The OER theory accounts for the difference in spatial ability between the two types of men in terms of $E_2$ levels. Furthermore, the theory explains relations between spatial ability and rate of body and brain maturation, menstrual changes, and alterations in steroid levels throughout life. Instead of being based on non-directional correlational data, the OER theory is built on experimental as well as descriptive data from normal as well as abnormal groups. What is more, the theory can be subjected to testing, and a number of quite specific predictions can be made on the basis of the theory. It can for example be expected that the pubertal decline in spatial ability of girls will be most pronounced in the highly feminine-looking girls, less conspicuous in girls intermediate in femininity, and absent in androgynous girls. In addition, $E_2$ values will be expected to show decreasing average levels across these three groups.

The OER theory is fully compatible with the idea of an impact of certain environmental factors on spatial ability. It is taken for granted that sensory stimulation is important for the development of perceptual processes generally. Other environmental factors such as nutrition, sleep, psychic stress, drug intake and physical activity can be incorporated by the OER theory in terms of their effects on $E_2$ and T levels (Kreuz *et al.*, 1972; Levin *et al.*, 1967; Rose *et al.*, 1969; West *et al.*, 1973). It is of interest, to test to what extent the OER theory can correctly predict the impact of specific environmental changes on spatial ability and perhaps thereby provide a "missing link" in the understanding of how social and other environmental factors influence the development of cognitive skills.

Spatial ability seems to be under genetic influence (Vandenberg, 1967) but apparently not in a straight-forward X-linked way. Perhaps spatial ability is coded for by one or more genes on an autosomal chromosome. The OER theory accounts for the role of genetic mechanisms in spatial ability by postulating that an autosomal gene for spatial ability is activated or inhibited as a function of the central concentration of $E_2$, according to the operon model of Jacob and Monod (1961). Thus, the OER theory considers spatial ability to be sex-limited and not sex-linked as proposed by Stafford (1961, 1963, 1972). The OER theory assumes that spatial ability is mediated at least in part by genes on an autosomal chromosome. However, intra-sex variability in spatial ability is larger than inter-sex variability, so sex-limitation probably cannot account fully for genetic influences on spatial ability. Accordingly, the OER theory also assumes that plasma steroid production is under genetic influence (Bock *et al.*, 1973; Garn *et al.*, 1969). Perhaps an X-linked gene exerts influences on steroid output, and thereby influences somatic differentiation and the expression of spatial ability by central induction of RNA and protein

synthesis, both during early brain growth and later by activational effects.

An important aspect of the OER theory relates to the origin of individual differences in spatial ability. The theory considers individual differences in spatial ability to be based on biochemical individuality. Because of this viewpoint, the OER theory represents a hypothetico-deductive model of inferences which makes it possible to generate a number of experimental hypotheses that can be rigorously tested. The emphasis given to individuality by the OER theory has consequences also for the traditional view on sex differences in spatial ability. The OER theory rejects the notion that high spatial ability is an exclusively masculine trait concomitant to the male karyotype. Instead, the theory approaches spatial ability on an individual basis. At present, the theory strives to relate spatial ability to the individual's central levels of $E_2$. In view of the fact that there is considerable overlap between the sexes with regard to peripheral levels of sex hormones, and since women and men show great individual differences in spatial ability with considerable overlap between the sexes, the OER theory advocates understanding spatial ability on an individual basis rather than on the basis of sex differences alone. According to the OER theory, it is therefore unacceptable to discourage women from entering areas traditionally dominated by men on the basis of the false notion that men are always superior to women in spatial ability, since some women are superior to most men in spatial tasks. Theories that fail to recognize the similarities as well as the differences between women and men in spatial ability fail to account adequately for available information of this pervasive aspect of behavior.

Evidently the OER theory is too simple. To give a few examples, the OER theory considers hormonal systems straightforward plus and minus systems, but the reality is that the immensely complex interactions of steroids and their intricate relations to neuro-transmitter substances are by and large unknown. The OER theory acknowledges later effects on target tissues of early steroid priming, although most details of such effects are not known. A cornerstone of the OER theory is the concept of central $E_2$ values, although there is as yet no safe method for determining these hypothetical values and further the assumed 1:1 relation between central and peripheral $E_2$ values is not proven. We are in great need of cross-disciplinary studies specifically designed for investigating various functional aspects of hormonal systems in addition to mapping covariant hormonal, neuronal, somatic, and cognitive development. The outcome of such studies undoubtedly will necessitate modifications of the OER theory or lead to the formulation of more adequate theories. But perhaps future studies better acknowledge already, from the beginning, the fascinating biochemical individuality characterizing human beings, and also that uncritical use of sex as *the* variable may be unduly restrictive. There are only four almost completely sex-dimorphic areas: menstruation, ejaculation, gestation, and lactation. All other human characteristics, and especially

psychological traits show far from complete sex-dimorphism. Studies not acknowledging this may be largely in vain.

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