He and She: The Sex Hormones And Behavior
By MAGGIE SCARF
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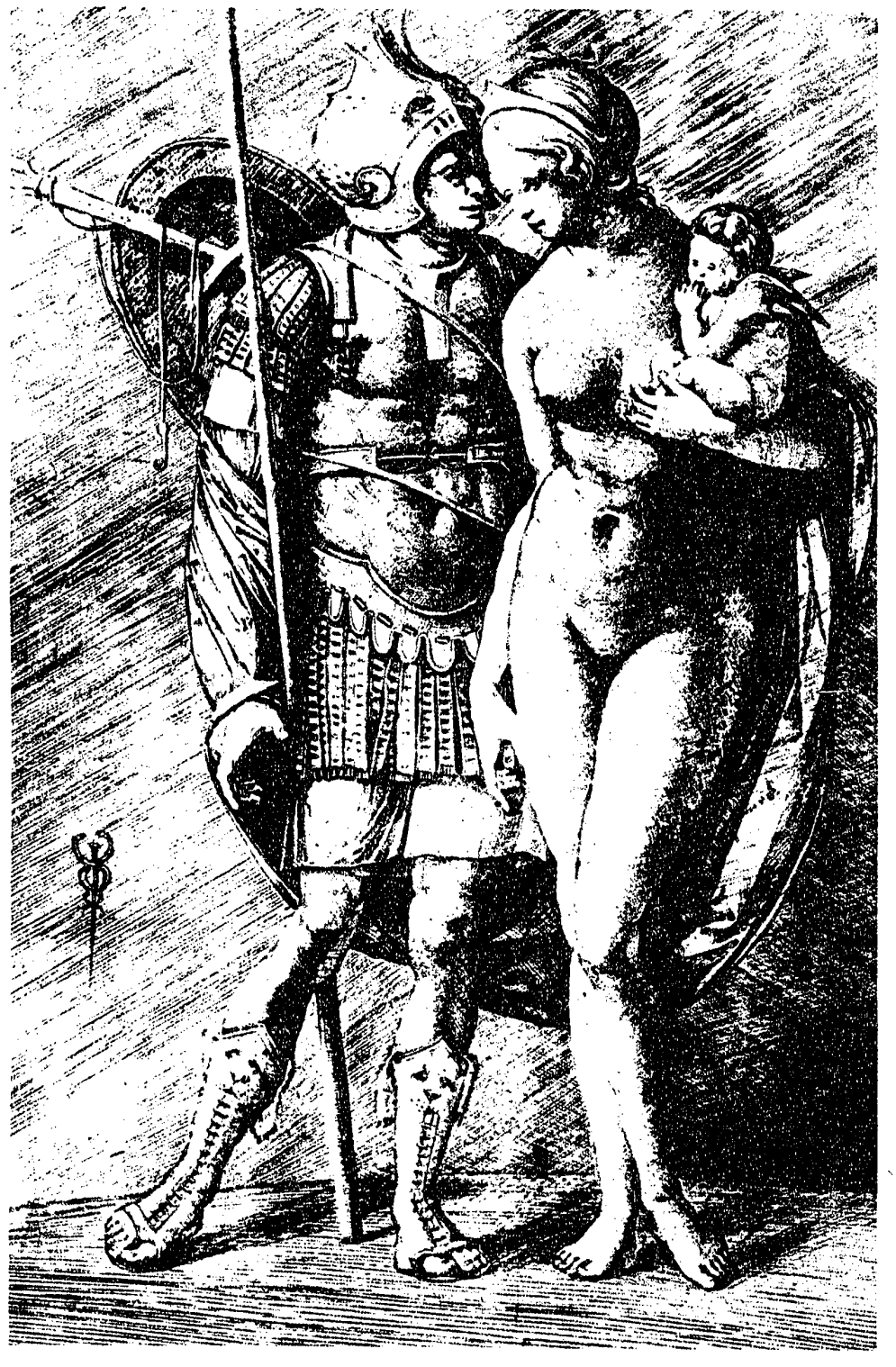

"Mars and Venus" by Jacopo de' Barbari.

FREUD always maintained that human psychology had, as one of its components, some unknown biological "bedrock." He thought that we all, male and female alike, were captives of our physiology: that inborn propensities and tendencies exerted a profound effect upon behavior—and that these inner propensities were different in the two sexes. (Hence his now-infamous remark: "Anatomy is Destiny.") This belief is, however, not popular in the present, more "environmentalist" intellectual climate. The common assumption nowadays appears to be that where male behavior and female behavior are different they are so because of acculturation: that the display of either "masculinity" or "femininity" is by and large the result of social training.

Recent research on the sex hormones suggests that it is Freud's ideas which may be the more valid approximation of the reality. Endocrine studies have now established the critical role played by the sex hormones during prenatal life: These hormones are not only crucial to differentiation of the (male or female) sexual organs; but they "program" the brain, during fetal development, for the later display of either masculine or feminine behavior.

The word *hormone*, in the Greek, means "to arouse"—and this is what hormones do. They are chemical substances, secreted first in one place (usually, but not always, a gland or organ), then released into the bloodstream to move through the body and exert their ultimate effects elsewhere—on other "target" organs. The hormones and hormone-producing glands are part of an interrelated chemical system, as intricately balanced as the body's "electrical" system (brain, spinal cord, nerves, sense organs). Hormones must be present in order for the initiation—or in some cases, inhibition—of a multitude of complex chemical processes. They are involved, for example, in the vital maintenance of correct blood sugar in the body; of the over-all rate of metabolism; in the regulation of water retention, of growth, of body responses to stress; and in the mediation of reproductive behavior.

The major sex hormones are secreted either in the testes in males (testosterone) or in the ovaries of females (progesterone and the estrogens, the important ones being 17 *beta*-estradiol and estrone). The adrenals, small yellowish organs lying just above each kidney, also secrete some sex hormones, including small amounts of testosterone and larger amounts of the weaker male hormone androstenedione (AD)—as well as a variety of other important hormones, including cortisol, cortisone and the "fight-or-flight" epinephrine (adrenalin).

Both sexes produce hormones of the opposite sex. In fact, men produce as much of the potent 17 *beta*-estradiol as adult women early in their menstrual cycle (when estrogen levels are at a low ebb). Men also have as much, or more, 17 *beta*-estradiol and estrone in their bloodstreams as do most postmenopausal women.

It is not the lack of estrogens which make a male a male, but the far higher levels of testosterone, antagonizing and nullifying the biological effects of the female hormones. An interesting little example of this is the recent finding that women after puberty have a greater sensitivity to odors than do men; and that this sensitivity is lost if male hormones are administered. (It is also lost when women are deprived of estrogen; and regained if the estrogen is replaced.)

IN both males *and females*, testosterone appears to be the hormone which most strongly influences levels of libido. Incongruous as this idea might seem in the case of the female, it is now well supported by documentation from many sources. Studies of women who have had their adrenals (where most of the male hormones produced by females are secreted) removed indicate that these women suffer a dramatic loss in sexual desire; women who have had their ovaries removed rarely respond to loss of estrogens with loss of sex drive. In a report on a group of women who had had both operations (adrenalectomy and ovariectomy) it was found that virtually all were affected postoperatively, some merely reducing the frequency of intercourse, some losing interest in sex entirely. A subgroup of the same patients, who had the ovariectomy earlier in a separate and prior operation, reported experiencing no change in sexual interest or desire at that point.

Studies of women receiving testosterone injections also confirm the current hypothesis that male hormone mediates libidinal drive in the female. In one survey of a group of women receiving massive male hormone dosages (in the treatment of breast cancer), it was found that 99 per cent of the patients were experiencing a marked surge in sexual desire. In another study, more than 100 women were treated with male hormone for such symptoms as

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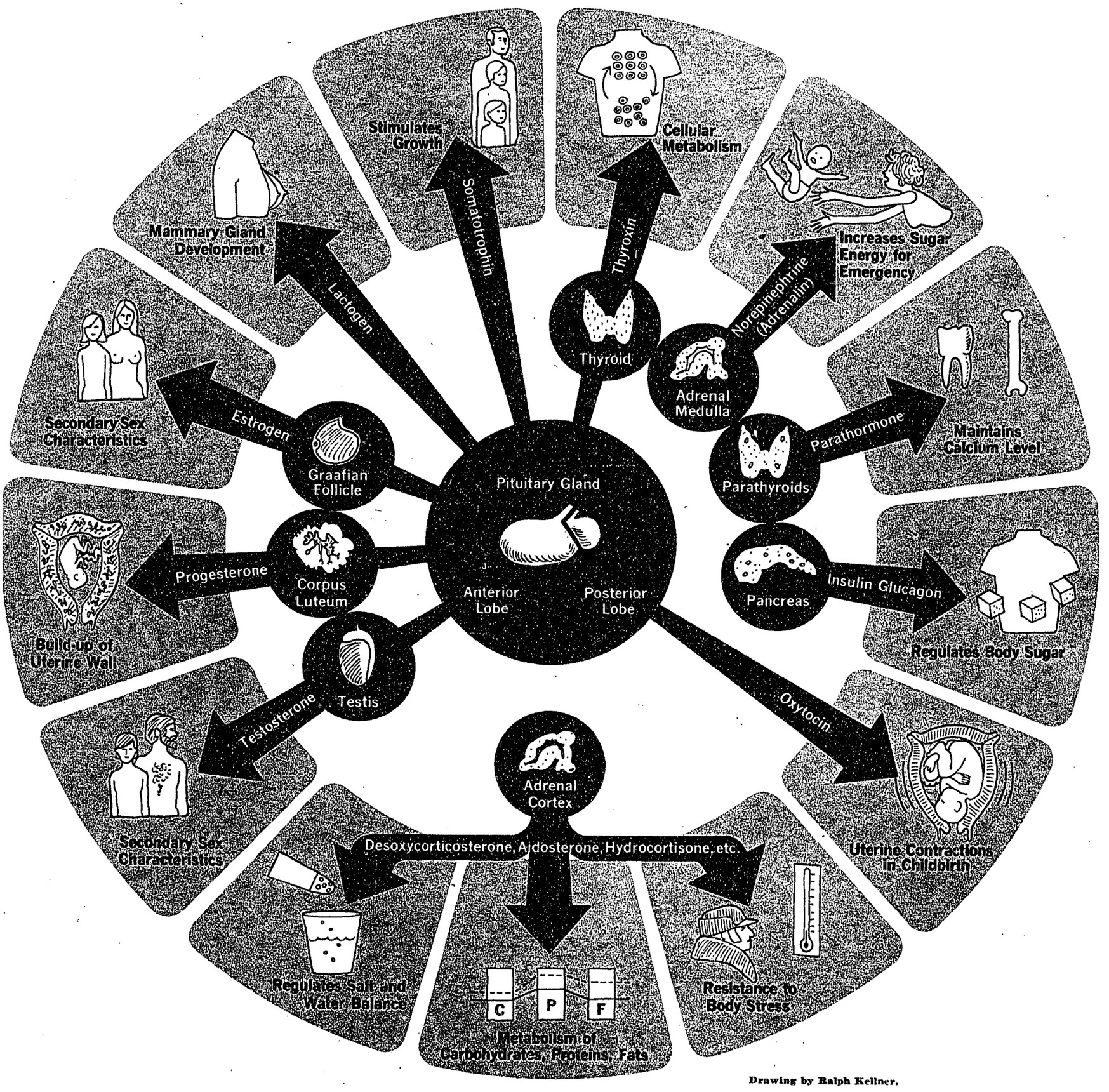
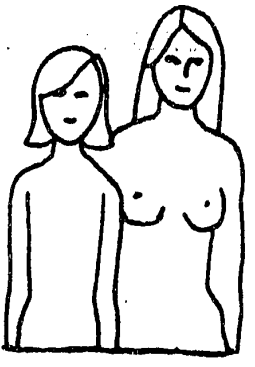


Drawing by Ralph Kellner.

Natural Chemistry. Hormones, secreted and released into the bloodstream, are crucial to the regulation of vital body processes, as shown in this chart adapted from an exhibit in the Hall of Man at the American Museum of Natural History. Recent research indicates that the interplay of sex hormones in the fetus not only determines sexuality, but may influence later masculine or feminine sexual behavior.

Sex hormones and behavior



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frigidity, dysmenorrhea, etc. More than three-fourths of these women responded with a reported rise in libido: some even complained that their sex drives had become excessive. (A small group of the same patients, treated for a period of time with estrogens, experienced no apparent change in libido whatsoever.)

In the male, testosterone has sometimes been used as a "maleness-bolstering" medication. Doctors have attempted to treat a variety of problems, including impotence, decreasing libido in aging men, and homosexuality with extracts of this most potent of male hormones. However, the efficacy of such treatments remains unclear. A massive review of efforts to counteract impotence with male hormones, published in 1947, concluded that physically healthy men did not respond to stepped-up dosages of testosterone; the problem was psychological. Where sexual performance did improve after male hormone injections, it was suggested, the improvement came from the easing of psychological anxieties.

A more recent (1970) research report suggests, however, that adding testosterone may after all have more than a placebo effect. In the study described, two groups of male patients with problems of impotence were compared: The men in the first group, receiving a placebo, showed improved sexual performance among less than half of their number. The second group, receiving real medication in the form of oral testosterone doses, responded with more adequate sexual functioning among more than three-quarters of the men taking part.

As to whether or not testosterone is useful in combating the sag in sexual drive sometimes experienced by aging males, the answer, simply, is not known—information on the results of such treatment is largely anecdotal and not, it should be added, particularly optimistic. Attempts to overcome homosexuality with added male hormones have, of course, been doomed: Testosterone influences libido strength in both sexes, but has nothing to do with determining the sex of the individual toward whom heightened sexual interest will be directed.

PUBERTY is a time when the sex hormones are said to be "awakening." The pituitary or "master gland" (an organ just under the brain, not much larger than a small pea) now begins sending increasing amounts of hormones called gonadotropins into the bloodstream. These are chemical messengers which, in the case of the male, stimulate sperm production and the secretion of testosterone by the cells of the testes. In the female the same gonadotropins (chemically identical to those of the male) are released by the pituitary; but in females they appear in sequence, rather than simultaneously. The first of these hormones stimulates the growth of the egg and its nest cells within the ovary, with an accompanying rise in estrogen secretion. The second gonadotropin, appearing slightly later in the cycle, subserves the production of progesterone, the female hormone which prepares the uterine lining to receive the fertilized egg. The sex hormones bring about, in their turn, the onset of secondary sexual characteristics—breast development in girls, growth of facial hair in boys, etc.—as well as the behavioral changes seen in adolescence.

Puberty, however, it now appears, does not constitute an "awakening" so much as it does a *reawakening*. Research during the past several decades has demonstrated that the sex hormones are, in fact, present during prenatal development. The concentrations in which they appear in utero are crucial not only to sexual differentiation (to produce a male or a female) but, it now appears, to differentiation of central nervous system tissues which will mediate masculine or feminine behavior during adult life.

The primitive gonad, . It should be mentioned here, is

sexually bipotential: it contains everything necessary for the fetus to develop either as male or female. There is a "rind," capable of becoming an ovary; a "core" which can develop as a testis; and two sets of internal duct systems, male and female. (One of them will become vestigial during sexual differentiation.) The "genital tubercle" grows into either a clitoris or a penis; the tissue above the urogenital groove either fuses, in the male, to become a scrotum or remains separate as the lips of the vagina.

What makes the embryonic gonad move toward differentiation as male or female? Surely it is genetic sex which sets a "direction"—and, it used to be assumed, determined everything that followed. But a series of brilliant experiments begun in the late forties by the French physiologist Alfred Jost gave definitive proof that it was in fact the prenatal hormones which played the decisive role in sexual differentiation of the developing fetus.

Jost, using surgical methods so delicate that they have been difficult for other investigators to imitate, castrated a male rabbit in utero. The infant male, when it was born, had completely *female* external genitalia: It appeared that in the absence of the testes (and therefore, testosterone) a genetic XY male fetus had developed in a female direction.

What would happen, then, to an ovariectomized female fetus? Jost removed the ovaries of a developing female rabbit fetus: At birth, she had normal female internal ducts and external genitalia. It seemed that the ovaries—and therefore, prenatal estrogens—were not vital to the female in order to ensure her normal differentiation. Indeed, given that no interference (such as the presence of testosterone) occurred, the fetus would always develop along female lines. Jost's work suggested that Nature had some fundamental bias in favor of producing females. Femaleness thus could not be—as Freud had suggested—some state of incompleted maleness; it appeared to be the basic form of life. Maleness was itself the correction: to achieve it, something had to be added on—male hormones.

(One psychoendocrinologist tells a story of how he explained to a very religious friend that the Adam and Eve story in Genesis was unlikely—that all biological evidence now available suggested that if one sex arose from the side of the other, it would have had to have been Adam who came from Eve. "Isn't God wonderful?" retorted his friend. "When He created the sexes, He even did it the hard way!")

Later work of Jost's, and a variety of other studies, have now demonstrated that testosterone must not only be present in utero in order for normal male differentiation to occur; it must be present during a sensitive "critical period." A male rabbit fetus, castrated by the 19th day after conception, will develop a completely female internal duct system and female genitalia. If castrated on day 24, however, when the crucial phase is over, its development will be completely male.

Similarly, a male rat castrated in utero (this can now be done using chemical methods) will differentiate in a female direction—with a vaginal pouch, unfused scrotal tissues and a miniaturized penis which is indistinguishable from a clitoris. If castration is delayed until the critical period has passed, however—in this species, several days before birth—the rat will be irreversibly male.

In females, the presence of

testosterone during the sensitive phase is as dramatic as its absence in males: A female rat receiving injections of male hormone during the critical period will become virilized, develop male-appearing genitalia, grow at an increased (male-type) rate, lose her reproductive cycle and become sterile. The same hormone doses, given 10 days after birth, will achieve none of these effects.

HORMONES—the right concentrations at the right times—are decisive to normal sexual differentiation. In the middle fifties, the group of researchers working with the great pioneer in hormones and behavior, Dr. William C. Young (who died in 1965), began to wonder: Was it possible that fetal hormones also had some determining effect upon the type of sexual behavior that would be shown much later on, at puberty? What actually caused males to show masculine sexual responses during mating, and females to display feminine responses? It had always been assumed that the reason, in each case, was genetic. A genetic male simply looked like a male and was expected to behave like one.

But if prenatal hormones could feminize his genitalia, and masculinize those of the female, could they also affect the two, complementary sets of behavior and the type of sexual responses each would show?

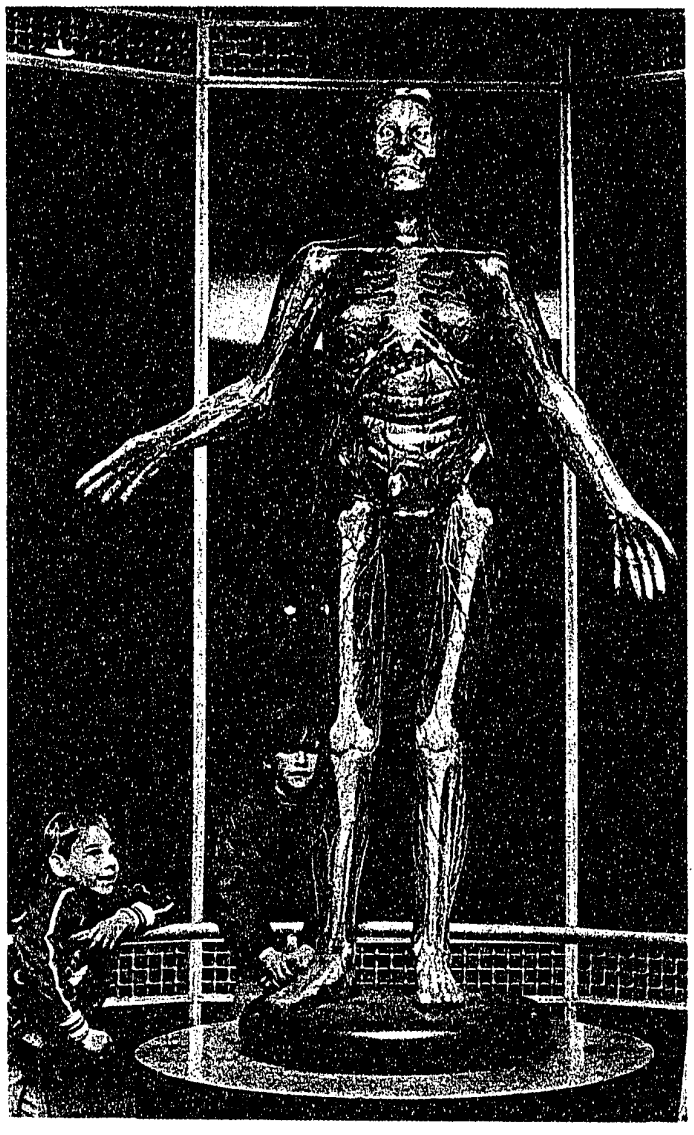
In a now-classic experiment, Young and his colleagues demonstrated that a female guinea pig which had been virilized during prenatal life (through testosterone shots to the mother) would, when given male hormones at puberty, respond with startling amounts of male behavior. In subsequent work, Dr. Arnold A. Gerall showed that such females would not only mount other females and display pelvic thrusting, but (granted that genital development had been sufficiently anomalous) even intromission and ejaculation. In contrast, even when given high doses of estrogens, the capacity for showing normal female behavior—such as the "lordotic" response, typical in female rats and guinea pigs, in which the back is deeply arched and the genitals raised and presented to the male—was dramatically diminished. It was as if, during the period of prenatal life, some inner behavioral dial had been set at "male."

Experimental studies of the past 10 years have now established that, at least in lower animals, there are sensitive neural tissues which (like the primitive gonad) are bisexual in potential. These tissues, located in the hypothalamic region at the base of the brain, differentiate during fetal development to produce an unequivocally "male" or "female" brain; that is, they become imprinted during prenatal life to mediate either masculine or feminine mating behavior at puberty. Again, the key to what happens is testosterone. If it is present, the "female" pattern will be suppressed and the "male" tissues will become organized for the steady release of gonadotropins at puberty, and for male sexual responses during reproductive behavior. If, on the other hand, testosterone is absent in uterine life, the sensitive brain areas will differentiate as "female." They will become programed for the cyclical release of pituitary hormones at puberty, and for female sexual responses during mating.

Might homosexuality in the male be tied to a less-than-adequate supply of testosterone during the critical period when brain tissues are differentiating and becoming "programed" for the display of later sexual behavior? A number of researchers, intrigued by a vast animal literature on the subject, have recently begun looking for a possible correlation between homosexual behavior and the actions of fetal hormones.

In a British report, published last fall, it was found that a group of homosexual males had lower levels of testosterone in their urine than did a comparison group of heterosexual males; and that a group of lesbian women had higher testosterone in urinary samples than did a control group of female heterosexuals.

This past year, in an investigation carried out at the Masters and Johnson research institute in St. Louis, the blood plasma testosterone values and sperm counts of 30 young homosexual college students were carefully analyzed. It was found that



WOMANKIND—The see-through woman at the American Museum of Natural History. Research now indicates that femaleness is not, à la Freud, an incompleted state of maleness. Instead, femaleness is the basic form of life, and maleness is a later "correction."

among the 15 men in the group who were totally, or almost totally, homosexual, testosterone readings were much lower than they were among the other half of the men, who had definite heterosexual proclivities also. Sperm scores were also astonishingly lower among exclusively homosexual males. There appeared, interestingly enough, to be no great difference either in hormone levels or sperm counts when the bisexual males were compared with a "control" group of heterosexuals. According to the director of this research project, Dr. Robert C. Kolodny, the important question to be studied now is whether diminished testosterone supply is somehow a *result* of homosexual behavior—or whether it reflects an endocrine makeup that is simply different from that of heterosexuals in the first place.

A fascinating addendum to the recent research on human homosexuality—and, certainly, food for speculation—is the work of Dr. Ingeborg Ward of Villanova University. In an experiment reported in the January issue of Science, Dr. Ward demonstrated that severe stress to a mother rat during pregnancy can block the normal behavioral development of her male offspring—and in effect, demasculinize them.

DR. WARD, trained as a psychologist, proceeded in this experiment by placing the rat mothers-to-be, periodically, into clear plastic tubes. The tubes, from which it was impossible to escape, were then illuminated from above by implacable, glaring lights. The animals responded with every sign of distress: urination, defecation, hair standing on end. (Rats fear these lights so greatly that, initially, when they were too bright, several of the animals died.) Other pregnant females, kept in a nearby vivarium, were not placed under stress, and served as control animals.

After birth, some of the male "pups" from the stressed mothers, and some from the unstressed mothers, were subjected to further adversities—they were placed in ice-cube trays which were shaken periodically on a vibrating metal rack. When the male offspring all reached the age of puberty (90 days in the rat), they were paired with females in heat.

Those males which had been stressed prenatally showed low degrees of masculine response, and little sexual interest (as did males which were both prenatally and postnatally stressed). They mated far less frequently than did the group which had been subjected to stress only after birth, or those which had not been stressed at all.

All of the rats where then castrated. Some 10 days later they were given injections of estrogen, which were followed up with shots of progesterone. The males were then paired with "stud" male partners. In this situation, those pups which had been stressed in utero displayed striking amounts of female sexual behavior, including the lordotic arch. The same high degree of feminine receptivity could not be elicited from males stressed after birth, or those not stressed at all.

It is Dr. Ward's belief that the nonmasculine behavior shown by the prenatally stressed pups resulted from abnormal neural tissue-imprinting during the critical period of fetal development. "In response to high degrees of environmental stress," she explains, "the pituitary began stimulating increased production of the adrenal 'stress' hormones. Included among these is a weak male sex hormone, androstenedione (AD). As a side effect of this situation, the testes also

MAN AND MONKEYS—Dr. Robert Rose of the Boston University School of Medicine, has used rhesus monkeys to show links between male hormones and aggression.

slowed down their production and release of the far more potent testosterone."

The weaker but more plentiful AD then competed with the more powerful testosterone, theorizes Dr. Ward, for control of the same chemical resting sites within the sensitive neural tissues—and the weaker AD won out. "The net result was that testosterone was unable to do its normal job of programing the brain," she says. "The tissues developed under the influence of the weaker hormone, and thus the animals were unable to differentiate as normally functioning males."

Dr. Ward is now trying to determine whether male rats will become feminized simply by being given large doses of AD prenatally. But at present the ingenious experiment described above stands as the sole demonstration that, by manipulating the prenatal environment, one can obtain exactly the same awesome alterations in male and female behavior as have been obtained previously only through direct manipulations of the fetal hormones.

ONE cannot of course generalize from rats to humans. (And the psychoendocrinological journals are as full of cautions about this temptation as the old temperance tracts once were about the dangers of drink.) Nevertheless, as one researcher remarked privately: "We do, in fact, work with the implicit assumption that what is found to be true in one species will hold true up and down the phylogenetic scale. It's usually an exception when one discovers a physiological mechanism in one species and then finds it absent—or totally reversed—in others. After all, aren't we making the same sorts of assumptions when we test out our drugs on rats?"

The presumption is, then, that the higher animals including monkeys, apes and human beings are, like the rat and the guinea pig, *not* psychosexually neutral at birth: That they are, even before the onset of learning and social experience, "programed" or predisposed by early hormonal influences to acquire specific, either masculine or feminine, patterns of behavior. In a study carried out in the late nineteen-sixties by Dr. Robert W. Goy, it was demonstrated that female rhesus monkeys, exposed to male sex hormones during prenatal development, would later behave in more male-like, than femalelike, fashion. Dr. Goy, working at the Oregon Regional Primate Research Center, injected a group of expectant monkey mothers with periodic doses of testosterone. The result was, not surprisingly, a generation of female offspring whose genitalia were male in appearance. These female "pseudohermaphrodites" were separated from their mothers at birth, and henceforth socialized only with their agemates.

Goy carefully studied the behavior of the virilized females as they grew into childhood. It had already been well established, through the famous monkey studies of Dr. Harry Harlow and others, that the play behavior of juvenile male monkeys was measurably different from that of the young females (and that these differences were not "taught" by the parent monkeys, because they manifested themselves even when the juveniles had no contact whatsoever with the older generation). The young males, for example, showed much more social threat behavior; they initiated play more often than did the young females; and they engaged in rough-and-tumble and pursuit play to a far greater degree. The males also withdrew less from threats and approaches made by others; and they engaged in more sexual play, including the frequent mounting which was in effect a "game" in which the future sexual role was being rehearsed.

The impressive thing about Goy's experimentally masculinized females was that they too behaved in all of these ways. They displayed the elevated levels of energy and activity commonly seen in young male monkeys: In fact their play behavior was much more similar to that of the male than to anything normally encountered in the behavior of the juvenile female.

In a 1967 study carried out

DR. ANKE EHRHARDT, whose studies show that humans' fetal hormonal history plays an important role in shaping later gender identity.

at the Psychohormonal Research Unit of Johns Hopkins Medical School, the same unusually high levels of energy and activity were found in a group of 10 young girls who had been accidentally masculinized in utero. This research investigation was carried out by Dr. Anke Ehrhardt, working in collaboration with the Psychohormonal Unit's well-known director, Dr. John Money. The 10 young females taking part had all been virilized as a result of what was essentially a medical mishap: Their mothers were given progestin, a synthetic hormone, during pregnancy (in order to prevent unwanted abortion). It was not known at the time—during the 1950's—that certain progestins have a masculanizing effect on the developing female fetus. Nine of the 10 girls had been born with male-like genitalia, including an enlarged clitoris and a fused, empty scrotum. They received surgical correction early in life, and development proceeded normally from that point onward; psychosexual development, carefully evaluated by Dr. Ehrhardt in extensive tests and interviews, was certainly within the normal female range also. But it did seem to point toward some interesting questions about what the influence of those masculinizing fetal hormones had been.

Of the 10 girls, ranging in age from almost 4 to almost 15, nine were out-and-out tomboys. They preferred trucks, guns and other boys' toys to dolls. They loved being outdoors, climbing trees, playing football and baseball. They preferred being with boys to being with other girls; they wore boys' clothing styles and were more or less indifferent — some were actively opposed—to skirts and more feminine modes of dress. All displayed a high frequency of self-assertion and self-reliance, some of them to such an extent that their mothers were concerned about their behavior. "My daughter acts like a boy," complained one woman. "It might be because of the hormones. She is the opposite from me. I was the dainty type." Another family was having problems because their fetally virilized daughter was far better in sports than was her older brother.

Says Dr. Ehrhardt, who is now an assistant research professor of pediatrics and psychiatry at the New York State University at Buffalo: "The girls were consistently less interested in doll-playing than were a 'control' group of 10 girls, who were matched with them in every possible way—age, race, socioeconomic level, I.Q., etc. Also, the 'control' girls did a great deal of bride-fantasying, and involved themselves frequently in those sorts of games which are actually childhood rehearsals of the future maternal and wifely roles. In contrast, the fetally masculinized girls tended to fantasize about future careers."

In studies which she and Dr. Money have done on girls suffering from adrenogenital syndrome, notes Dr. Ehrhardt, the same tomboyish element and high-energy level regularly appear. Adrenogenital syndrome is a genetically transmitted condition which causes masculinization of the female fetus during prenatal development. The condition is due, briefly, to an error in metabolism which causes the adrenals to become overactive and produce too many hormones, including too many male hormones. It is now possible to stabilize this dysfunction with cortisone, so that overproduction of male hormones in the adrenogenital girl can be stopped postnatally, and her genitals can be surgically feminized. Still, psychosexual development of these girls, similarly to the progestin-induced masculinized girls, is toward the more "malelike" end of the normal female spectrum—high degrees of activity expressed in more masculine kinds of behavior.

IN assessing which behaviors were to be called "masculine" and which were to be called "feminine," Drs. Ehrhardt and Money relied on criteria such as energy expenditure (much higher in boys), toy and sports preferences, career ambitions, maternalism (girls are usually fascinated by infants and infant care; boys are usually not) and several other items, including body image, clothing choice, etc. In statistical analyses of responses of large groups of boys and girls, sex-related "male" and "female" clusters about these items do reliably emerge.

"Nevertheless, isn't it possible," I asked Dr. Ehrhardt, who is a fair-haired, pretty German-born woman in her early 30's, "that these 'sex differences' are merely artifacts of our culture? Most psychiatrists and psychologists (and of course, most Women's Liberationists) believe that they result primarily from social experience. That is, a small female child is taught very early, or learns by imitation, those 'feminine' ways in which she is expected to behave—and responds by doing it."

"I would agree," she answered, "that the most powerful factors in the shaping of gender identity are probably

DR. INGEBORG WARD in her Villanova University laboratory. Her research has shown that a mother rat under stress during pregnancy can demasculinize her male offspring; a scientific debate has ensued over whether such results are applicable to human homosexuality.

experiential and social. In other words the primary thing is whether a person is called and thought of (and calls himself or herself) male or female. This is of course fundamental to identity. But within the broad spectrums of behavior which we call either masculine or feminine, there are certainly very wide variations. You can have, on the one hand, a woman who is totally domestic and maternal; and on the other, a person who is uninterested in children and wants only a career. My speculation would be that there is a fetal hormonal history, in both these cases, disposing the individual in one direction or the other. In other words, what I'm suggesting is that there may very well be normal female hormone correlates to the variations of normal female behavior.

"The main message of most of this work, both with animals and with humans," she added, "is that hormones before birth may have an organizing effect upon behavior that will appear only much later—that social environment is the mold in which basic tendencies, already present, will be shaped and formed. The idea is that testosterone, by its presence or absence, sets some kind of behavioral potential; and that postnatal experiences are actually acting upon a physiologically biased substrate."

One very strange factor emerging from Dr. Ehrhardt's study of the 10 progestin-virilized girls was that their I.Q.'s were all unusually high. Six of them had I.Q.'s above 130; thus, in a random sample in which one would normally expect to see this elevated value in 2.2 per cent of cases, it appeared in 60 per cent. In an earlier study of 70 adrenogenital girls and boys carried out by Drs. Money and V. Lewis, the same peculiar incidence of high I.Q. was encountered. In a group where, it would have been expected, 25 per cent of those tested would have I.Q.'s above 110, it was found that there was an actual observed frequency of 60 per cent having I.Q.'s above that value.

"DOES this make you think," I asked Dr. Ehrhardt, "that boys, who are normally exposed to more masculinizing hormones in utero, would be expected to be ipso facto brighter than girls?"

"I don't think boys are brighter," she answered quickly. "But again, female and male intelligences do tend to cluster, statistically, around different sets of abilities. Boys appear to do better in mathematics and more abstract kinds of intellectual functioning, while girls score much higher in verbal capabilities." She paused: "I would be willing to allow that chemical influences in prenatal life might increase the level of energy and activity; and that they might have some enhancing effect upon intellectual capacities."

In a recent British publication, "Antenatal Progesterone and Intelligence," by Dr. Katherina Dalton, the very same phenomenon appeared. Dr. Dalton studied a group of boys and girls whose mothers had been given progesterone during pregnancy. (Progesterone, a female hormone, is similar to the progestins, but has no masculinizing side-effects.) All of the progesterone offspring studied by Dr. Dalton, both male and female, progressed better and faster than a comparison control group of children. They stood earlier, walked earlier, received significantly better grades in academic subjects, verbal reasoning, English, etc. Moreover, the more of the hormone their mothers had received, the earlier they walked and the better they did in school.

Thus, according to Dr. Ehrhardt, the intelligence-enhancing effect, if it proves to be definitely there, may have nothing to do with the masculinizing effect: "The kids in Dr. Dalton's study showed an increase in I.Q. without becoming virilized. So what we're seeing may just be due to some general chemical influence of these hormones. As far as I'm concerned, the whole question of the connection between intelligence and prenatal hormones is definitely a wide-open one." ■