

medical classifications of intersex

from Anne Fausto-Sterling, "The Five Sexes," and the Intersex Society of North America website (isna.org)

1) based on anatomy of gonads and genitalia:

herms: so-called true hermaphrodites, who possess one testis and one ovary in same organ
merms: male pseudohermaphrodites, XYs with testes, vagina, and clitoris (do not menstruate)

ferms: female pseudohermaphrodites, XXs who have ovaries and aspects of male genitals

2) based on hormones:

Progestin-induced androgenization

Caused by prenatal exposure to exogenous androgens, most commonly progestin. Progestin is a drug that was administered to prevent miscarriage in the 50's and 60's and it is converted to an androgen (virilizing hormone) by the prenatal XX person's metabolism. If the timing is right, the genital anlagen is virilized with effects ranging from enlarged clitoris to the development of a complete phallus and the fusing of the labia. Virilization only occurs prenatally and the endocrinological functionality is unchanged, i.e. feminizing puberty occurs due to normally functioning ovaries. In other words, XX people affected in-utero by virilizing hormones can be born into a continuum of sex phenotype ranging from "normal female with large clitoris" to "normal male with no testes." NB: use of progestin is not effective in preventing miscarriage.

Congenital Adrenal Hyperplasia

Caused when an anomaly of adrenal function (21-hydroxylase or 11-hydroxylase deficiency) triggers the synthesis and excretion of an androgen precursor, initiating virilization of an XX person in-utero. Because the virilization originates metabolically, masculinizing effects continue after birth. As in PIA, sex phenotype varies along the same continuum, with the possible added complication of metabolic problems that upset serum sodium balance. The metabolic effects of CAH can be counteracted with cortisone. The scenario for medical intervention for intersex is similar but CAH people have an increased likelihood of early detection due to metabolic imbalances.

Androgen Insensitivity/Partial Insensitivity Syndrome

In AIS/PAIS the cellular metabolism of an XY person is such that the cells do not respond to the effects of androgens. Endocrinological function is normal... but the cell's ability to bind androgens, due to compromised receptor site metabolism, causes a partial or complete lack of response to virilization. PAIS produces effects similar to CAH or PIA in a neonate with genital ambiguity. With complete AIS a neonate shows no indication that they are intersexed as the external genitalia are completely phenotypically female. Internal female structures do not develop, however, because Mullerian Inhibiting Hormone is present and prevents the formation of a female genital tract (oviducts, cervix, uterus, part of the vagina). Another form of AIS is present in individuals with 5-alpha reductase deficiency. During the formation of the male genital tract from the Wolffian duct portion of the genital anlagen, the target tissues

are not responsive to testosterone, [so] another form, androstenedione is required at this stage. The required enzyme, 5-alpha reductase, is missing so these people may be assigned and reared as girls. However, since pubertal genital tissue is sensitive to the effects of testosterone... such a child could experience masculinizing puberty and genital growth — assuming that their gonads have not been removed.

Turner's Syndrome Children are born with an XO karyotype and, in the absence of gonads, develop without any endocrinological influencing sexual phenotype. What this means, however, is that they are phenotypically female and raised as such.

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