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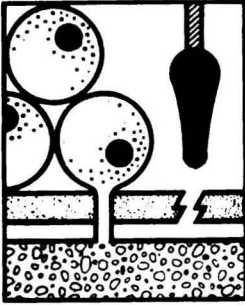
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MALE FERTILITY REGULATION WITH CYPROTERONE ACETATE (CPA)

L. Moltz, U. Koch, U. Schwartz, A. Rommler & J. Hammerstein

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Medium dose CPA (10-20 mg daily, p.o.) was administered to 30 fertile men for 12-26 weeks. Sperm analyses and hormonal measurements were performed before, during and after CPA medication. CPA decreases sperm count, density, and motility. A rise of pathological, immature and dead spermatozoa was observed. CPA also reduced *in vitro* and *in vivo* sperm migration. Glycerol-phosphorylcholine and acid phosphatase in seminal plasma were diminished, while alkaline phosphatase was elevated. Fructose and sialic acid were not changed. CPA significantly suppressed basal LH and FSH as well as pituitary responsiveness to GnRH; the episodic fluctuations of peripheral gonadotropins remained unaffected. Peripheral testosterone and dihydrotestosterone decreased significantly; the pulsatile pattern of androgen secretion was abolished. The majority of these alterations did not become evident until weeks 15-18 of CPA administration. All changes were completely reversible. No serious clinical side effects including loss of libido occurred. One conception occurred 10 weeks after antiandrogen treatment. CPA exerts an inhibitory effect on several parameters of male fertility. The contraceptive efficacy could not be determined since this was a phase I clinical trial. All pregnancies, despite CPA medication, occurred within the first 4 months of drug intake.

EFFECT OF LOW DOSE OF ALPHA CHLOROHYDRIN ON HISTOCHEMICAL & BIOCHEMICAL CHARACTERISTICS IN RAT TESTIS & EPIDIDYMIS

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It is unknown whether alpha chlorohydrin affects the spermatozoa directly or induces the spermatozoal sterility by testicular and epididymal dysfunctions. Histochemical and biochemical studies were made to evaluate effects of a low dose (6.5 mg/kg/9 days) and high dose (140 mg/kg) of alpha chlorohydrin on the rat testis and epididymis. Low dose of alpha chlorohydrin did not cause any appreciable histological change in testis and epididymis but high dose caused degenerative changes. However, low dose caused conspicuous histochemical changes in various cell types of testis and epididymis, which were extended and confirmed with biochemical data. Several biochemical parameters decreased in various cell types of testis and epididymis, phospholipids, RNA, protein, glycogen, ATPase, 5'-nucleotidase, hexokinase, phosphoglucoisomerase, phosphofructokinase, aldolase, triose phosphate isomerase, glyceraldehyde phosphate dehydrogenase, phosphoglycerate kinase, phosphoglycerate mutase, enolase, pyruvate kinase, lactate dehydrogenase, alcohol dehydrogenase, isocitrate dehydrogenase, malate dehydrogenase, succinic dehydrogenase, NAD diaphorase, glutamate dehydrogenase, hyaluronidase, lipase, non-specific esterase, and Y-glutamyl transpeptidase. But triglycerides esterified cholesterol, alkaline phosphatase, glucose-6-phosphatase, phosphorylase, glycerol-phosphate dehydrogenase, RNAase, and proteinase were increased. The chemical constituents which showed a decrease only in the epididymal epithelial cells and not in testicular cells, included sialic acid, glucose-6-phosphate dehydrogenase, 6-phosphogluconate dehydrogenase and NADP diaphorase. The components, which increased only in the cells of epididymis, were B-glucuronidase, β -glycosidase and sialidase enzyme. The biochemical constituents, which did not show any change in the cells of testis and epididymis after administration of alpha chlorohydrin, were DNA, glycerol, acid phosphatase, Δ^5 -3 β -hydroxysteroid dehydrogenase and DNAase. It seems that alpha chlorohydrin does not affect sperm metabolism directly but probably by altering the metabolism of testis and epididymis (especially of its epithelial lining).