

amounts of hormones usually produces a so-called "compensatory atrophy" in the specific gland in question.

To illustrate to you how this works, I would like to describe a built-in human experiment which was reported by Dr. Cahill, a famous New York City surgeon, a number of years ago when he first showed, and reported, that in patients having a one-sided carcinoma or cancer of the adrenal gland with hypersecretion of that gland, that the contralateral adrenal gland in all cases was almost completely atrophied. After surgical removal of the gland containing the carcinoma the patient succumbed to an adrenal insufficiency. At that time we did not have adequate therapy for this condition. As an adjunct to this, when patients are given cortisone in large doses for arthritis, asthma, or to suppress adreno-cortical secretion in carcinoma of the breast, it has been found on examining those deceased that adreno-cortical atrophy was maximal after only 15 to 20 weeks of continuous administration of only moderate doses of cortisone.

And now we will take a look at the male counterpart of the ovaries, the testicles; both of these glands, or gonads as they are termed, are derived from the same original source of cells. As early as 1948 it was demonstrated by Dr. Heller and Dr. Warren Nelson in a group of normal young adult males that the administration of the male hormone in large doses produced a marked and nearly complete destruction of the tubular elements of the testicles with a complete lack of sperm.

At the end of 6 months following the cessation of the administration of male hormone only a partial degree of recovery was noticeable. Even after 17 months the recovery was not 100 percent complete. In two studies which have been made by pathologists on the ovaries from patients treated with synthetic oral contraceptives, markedly abnormal changes were found in these glands, and they have been reported in the literature. In contrast to the smaller, greyish-white ovaries of the women on synthetic oral contraceptives are the normal appearing ovaries noted at cesarean section on women who have been under constant and very high levels of their own hormones as well as those produced by the placenta and fetus.

Mr. DUFFY. May I just clarify your position on these earlier studies that you say were wrong. It is your contention that there is no increase in fertility after ceasing to use oral contraceptives?

Dr. WHITELAW. Just the reverse.

Mr. DUFFY. The only question I would have then is: your study was completed about 1959, which is now some 11 years ago. Is there any possibility that these studies and their results which you dispute could be later studies that have gone into this in much greater detail or used more advanced techniques which were not available to you at the time you conducted your studies?

Dr. WHITELAW. Well, nobody else has made a study, as far as I am aware except for Dr. Sharman's corroborative findings. As I stated before, the whole basis of this premise is false to begin with. The only reason the oral contraceptives were given to these women was in the hope that there would be a so-called rebound. These women had normal ovulation previous to the time that they were given the oral contraceptives, so what the logic behind this is is not known to me. It was put on the basis that they rebounded. Well, they only rebounded