got atrophy and, therefore, why not by the same thing in women who already had hyperplasia.

In subsequent clinical studies Kistner reported these effects in detail. (Kistner, R. W., Histological effects of progestins on hyperplasia and carcinoma in situ of the endometrium, Cancer: 12: 1106, 1959, and Kistner, R. W., Treatment of carcinoma in situ of the endometrium, Clin. Obst. & Gynec. 5: 1166, 1962). These reports indicated that synthetic progestins produced marked changes in the glands and stroma (these are the two cellular types present in the lining of the womb) of adenomatous hyperplasia or carcinoma in situ. Profound gland atrophy and conversion of the stroma, the supporting network, to decidua were constant findings following prolonged therapy with these compounds. During the interval 1958–69, over 200 patients with either adenomatous hyperplasia or carcinoma in situ have been treated by progestational agents. In no instance did subsequent invasive carcinoma occur in the patients who were treated prospectively. These observations have been confirmed by numerous other investigators.

The final step in the progressive study of the effects of synthetic progestins on endometrial abnormalities was to test the efficiency of these agents on widespread, metastatic cancer of the endometrium. Kelly and Baker, Kistner, Anderson, Truscott, and numerous other investigators have reported an objective, not subjective, remission rate of 30 to 35 percent in such patients with some remissions lasting up to 6 years. The synthetic progestins, Delalutin and Depo-Provera are now generally accepted as optimum chemotherapeutic agents in the treatment of endometrial cancer when it is widespread. The mechanism of action is unknown but local effect of the progestin on estrogen binding sites in endometrial cancer cells has been suggested by the reports of Kistner and Griffith (Clin. Obst. & Gynec. 11:439, 1968) and Varga and Henriksen (Obst. & Gynec. 18:658, 1961). Added support for a local effect of progesterone on the endometrial cancer cell has been furnished by the report of Nordquist who cultured specimens from 11 uterine adenocarcinomas on Gelfoam in human serum. Addition of progesterone to the culture produced total necrosis in nine tumors. The addition of male hormone (androsterone) was less effective and cultures to which estrogen was added showed only slight alteration.

As a result of these and other studies, I have stated and I quote "Those patients who were destined to develop endometrial cancer from, or in, areas of hyperplasia will not develop that cancer if the oral contraceptive prevents the development of, or causes regression in, a typical endometrial hyperplasia."

Are there any questions?

Senator NELSON. Thank you, Doctor. Senator McIntyre.

Senator MCINTYRE. Dr. Kistner, I hope you will not take this too personally but since minority counsel felt it was necessary to question some of the witnesses yesterday about the capacity in which they were testifying here and whether or not they had any financial interest in contraceptive products that are competitive with the pill, I think it is only fair that we ask some similar questions of you in order to place your testimony in proper perspective.

Doctor, are you testifying only as an individual or are you testifying on behalf of or at the request of someone else?