from her physician, drug package inserts, and other reading materials. Unfortunately, I would agree with Dr. Connell that public pronouncements on the pill, either pro or con, are known more for their rhetoric than for their objectivity, often leaving both physician and patient misinformed or confused.

Acting as chairman of a department of obstetrics and gynecology, I can testify to the fact that many physicians are as confused as their patients in this respect.

Since these hearings will aid in the dissemination of information, I appreciate the opportunity to present a perspective on the pharmacological properties of the medications. That is what I would like to talk about now, what we know about some of these oral agents, what we can predict about the side effects, and so on.

Most oral contraceptives, whether of the sequential or combined type, contain a synthetic estrogen used in sequence or in combination with a synthetic progestin. I will describe each in turn.

Synthetic estrogens have been available since 1937 when Dodds first described diethylstilbesterol which is a synthetic as potent as the natural estrogen, estradiol, but unlike the native hormone is active by mouth. Since then, two other synthetic orally active estrogens have been produced—ethinyl estradiol and mestranol. I bring these up because these are the two compounds which are used in most of the contraceptives. They are of almost equivalent potency to one another but some 10 to 25 times as active as diethylstilbesterol. The latter two compounds are the ones most frequently used in contraceptive medication.

All of these active estrogens, whether it be estradiol or mestranol, have a comparable spectrum of biological properties and like the native hormone have actions on the reproductive tract as well as broad metabolic effects. The observation written in the FDA report and publicized that contraceptive pills containing these estrogens have extensive metabolic effects on many tissues of the body is neither novel nor surprising to any student of estrogen physiology. This is a normal consequence of estrogen action, and no particularly unique activity, of any of the synthetic estrogens has yet been described.

In any case, synthetic estrogens have been in extensive clinical use for 30 years. Very high doses have been used in men with prostatic cancer, for instance. There has never been a documented case of induction of cancer in the human by these agents and no serious sequelae have been reported save for their current implication in the embolism and phlebitis associated with pill usage and this is reported with a high dosage. If you recall, the British experience and the recent American experience, that thromboembolism seems to be less frequent in the patients taking the smaller dose, the preparation having the smaller dose estrogen.

I notice that you have the book on the metabolic effects of these contraceptives in the human and I know it says in there that you cannot equate one drug to another, nor can you equate it to the native hormone. But I know of no information that indicates that the biological properties of the estrogens used in the contraceptive pill are any different than stilbesterol for which we have at least 30