

no pill alternative. I submit to you, since most of the time we have no idea what causes idiopathic thromboembolic disease, it is not possible to carry out accurate matching.

As a matter of fact, the most important of the studies, the Vessey and Doll study—where they interviewed the patients, and went through the whole classical retrospective approach, and subjected their data to standard statistical tests—after they did all their matching, it turned out that the frequency of previous thromboembolic disease in one group was different from that of the other.

Now, the adviser to the Vessey-Doll study himself once said, in another statistical study, that if you find that an important factor is not adequately compensated in the matching process, then the whole study is invalidated; and I can give you that reference if you wish.

Therefore, I believe that what they did, which was to say, "Now, we will adjust our data and we will take only those sets that are now rematched" is not a valid, statistically acceptable way to study this problem; I submit that there are statisticians who would not accept the results of this study because it required post-data adjustment.

I don't wish to go into further statistical debates. I have discussed this particular study with Dr. Cochran of Harvard, Dr. Batson, and others, and they are all uneasy about the report.

What is more important is coming back to the question of bias. It turns out from a recent publication from the British literature that English women who take pills are heavier smokers than women who do not take pills; and smoking is a known contributory cause to thromboembolic disease.

Therefore, the question now arises, how much of the increased incidence of thromboembolic disease attributed to the pills is actually due to the fact that the pill takers in the British study were heavier smokers? I don't know the answer to that, but it does raise the question of how solidly one can accept the conclusion.

I want to make one further comment on the British study. Vessey and Doll, in print—and every other statistician will agree—clearly admit the fact that a study of this kind does not demonstrate a cause and effect relationship. It demonstrates only probabilities of association. Yet what has happened is, from the original statement that this does not establish a cause and effect relationship, we have crept by successive increments of not entirely convincing data to the point where we say this causal relationship is an established fact.

Now let us consider Sartwell's study, which has a considerably better design, but which, in the view of some statisticians, also has drawbacks. One of them, again, is the problem of the matching and the internal consistency. I show you here their own breakdown from their table 8, of the statistical risk of thrombophlebitis in different cities.

Senator McINTYRE. What page are you on?

Dr. GOLDZIEHER. Page 10.

Senator McINTYRE. I have it.

Dr. GOLDZIEHER. In their table 8, the data from Baltimore, Washington and Pittsburgh did not achieve statistical significance as far as an increased risk of thrombophlebitis was concerned. In New York, the difference of a factor of four was significant, and in Philadelphia a factor of 17 was highly significant.