reasonable. Rates for Comparisons 2 and 3, however, particularly the latter, have large variances because they are linear combinations of several estimates of annual per cent changes in mortality. Comparisons 1 and 4 may, therefore, be more useful indicators. The expected value used in the final summary statistic, mean per cent change in mortality rate (Figure 1), puts greatest weight on mortality for women during 1957-1961, less on mortality for men during 1962-1966, and still less on mortality for women during 1951-1956, and men during 1957-1961.

We used the per cent changes in mortality in comparison groups as measures of expected per cent changes in mortality for women in the reproductive ages during 1962-1966. That is, the assumption was made that the effects of most factors that influence mortality trends, such as diagnostic fashions, are proportional to the level of mortality. Vessey and Weatherall[11] made essentially the same assumption in their analysis of mortality in England and Wales when they extended straight regression lines plotted on a logarithmic scale from rates that were observed in the years prior to the introduction of the oral contraceptives.

In addition to the four comparisons incorporated in the derived statistics, two less formal contrasts provide evidence about whether the mortality changes for women in 1962-1966 are consistent with the pattern of oral contraceptive usage in the United States. First, any excess mortality from oral contraceptive use should be confined to the reproductive ages, although concurrent increases in hormone therapy for menopausal symptoms may make this pattern less precise. Second, the greater use of oral contraceptives among the white suggests that the impact on mortality should be greater for this portion of the US population.

On the basis of the above criteria, we believe the results indicate an association of the oral contraceptives with an increase in mortality from ICD category 460-8, but not from categories 332 or 420. The positive association for 460-8 is generally true for all four comparison groups. The relative increases, furthermore, occur to only a minor extent outside the childbearing years of age, particularly in the white population.

In contrast, the results for categories 332 and 420 do not meet the criteria we have set for a positive association with oral contraceptive use. For 332, the only age group among whites with increased mortality by all four comparisons was 25-29. Consistently elevated mortality was also found for this age group of nonwhite, where the next older age group was also consistently positive. The negative results in the white groups 30-44, given the limitations of the data and analytic method, suggest that there has been no effect of oral contraceptives on cerebral thrombosis mortality. We do not feel that our analysis has sufficient power to conclude that there has been an effect confined to the 25-29 or 25-34 age groups.

The data for category 420 are equivocal; although there are several small to moderate relative increases in both white and nonwhite reproductive age groups, they are neither so large nor so consistent as those occurring in category 460-8.

A relation of oral contraceptives to venous thromboembolism, but not to cerebral or coronary thrombosis, may derive from basic differences between venous and arterial circulation. Thrombosis and embolism in rapid circulation is probably determined by a different set of factors from thrombosis in the veins of the lower extremities, where varicosities, trauma, pregnancy, infection, and the degree and nature of physical activity are fairly well recognized factors.

Extensive publicity on adverse effects of oral contraceptives may have led