This is known as a "frequency polygon." If the number of measurements is large, it will not be difficult to draw a continuous curve through the angles of the polygon. Now, as a matter of fact, in the majority of cases of measurements of one detail, as, for instance, of the length of one or other organ or part of an organ in a homogeneous population, this curve of variation approaches curiously close to what has long been known to mathematicians as the curve of "probability of error." And if we venture to assume that these curves are identical, then we have an exact mathematical basis for our study of inheritance and its effects, for this curve of probability of error is a pure curve, and as such its various properties can be studied and determined. It is this assumption which Professor Pearson has made, and, starting from this, he has already obtained most valuable results. He is able to obtain such values as the probable error, probable deviation, the mean (or abscissa of the "centre of gravity" of the area enclosed by the frequency curve), the mode (or abscissa of the ordinate of maximum frequency), the standard variation (or index of variability)—and gains thus a valuable basis for discussing asymmetrical frequency or skewness.

For he finds that in biological statistics most frequency polygons are not symmetrical about the maxim ordinate. This skewness may be due to one of two sets of causes:

(*a*) The material dealt with may be heterogeneous and consist of a mixture of two or more homogeneous materials.

(*b*) The material may be homogeneous, but tend to deviate more to one side of the mean than to the other.

I will leave those of you who are interested in the matter to follow the mathematics of the subject, clearly set forth as they are, in Mr. Worthington's paper. What I wish to point out is that by the application of Karl Pearson's methods to the material in the hands of the great insurance companies, it will be possible to give a mathematical expression to the effects, if any, of parental disease upon the duration of life of the offspring by comparing the frequency curves of death of those of tuberculous ancestry with the frequency curves of the deaths of those of non-tuberculous ancestry. It will further be possible to determine whether there is such skewness or asymmetry in the first curve as can best be explained by assuming that we have a mixture of two or more homogeneous materials, *i.e.*, whether one group of the offspring are strengthened, and so afforded a longer period by parental tuberculosis, another definitely weakened and led to die at an earlier age by the same influence. It will also determine the value to be placed upon the influences of family tuberculosis when the parents themselves are not tuberculous.