by an increase in the rate of respiration and an even more marked increase in the depth of the individual respirations.

With these facts as a basis it was possible to analyze the factors present in cardiac disease which led to the production of dyspnea. It was well known that the tendency to dyspnea and the intensity of it when present presented great variations in different cases of cardiac disease, and it might be anticipated that the conditions and causes leading to its development would be different. The first of these factors to be studied was that of the metabolism.

Cases were studied in the Sage calorimeter and by means of the method of respiration calorimetry, the results in general being the same. It was found that the cases could be grouped according to the state of metabolism. In the first group were those cases in which this was within normal limits, and in this group were found all of the cases of cardiac disease which were well compensated. In the second group there was found to be more or less increase in the metabolism and this group contained those cases which showed more or less failure of compensation. Not all of the noncompensated cases, however, showed an increase in metabolism. Thus metabolism alone as a factor in the production of dyspnea in heart disease proved to be a variable feature.

The minute volume of the respiration was then studied and again the well compensated cases gave results within the normal limits. In cases of severe heart disease with a tendency to dyspnea the minute volume was found to be high. In all cases the minute volume was taken while the patients were at rest. It was found that the changes in this factor bore no relation to the state of the individual's metabolism. This high minute volume was proved, therefore, to be a factor of considerable importance in the production of cardiac dyspnea, for if the minute volume at rest was high it left a much restricted range for increase under conditions of exertion, even if mild.

A few years ago the occurrence of acidosis in heart disease was brought forward and used to account for the occurrence of dyspnea in practically all cases. It was proved that the respiratory centre was excessively sensitive to changes in the reaction of the blood in the direction of a reduction of its alkalinity. More extensive study of this factor was undertaken and it was shown that in cases of pure cardiac disease in a state of compensation there was no increase in the hydrogen ion concentration of the blood. In cases of pure cardiac disease with acute loss of compensation this factor was found to be variable, although in many there was found to be some increased accumulation of carbon dioxide in the blood, probably due to factors in the lungs which interfered with its diffusion. These studies proved, however, that carbon