is one of very great importance. I recently saw a case of typhoid fever where a fatal result came about indirectly, if not in a measure directly, from heart dilatation consequent on a secondary infection.

A word more about the symptoms and physical signs of cardiac hypertrophy.

The recognition of cardiac hypertrophy is easy when it is well marked, but difficult, if not impossible, when slight. There is seldom any pronounced subjective symptoms while the cardiac muscle retains its normal vigor. It is only when degenerative changes have set in that the patient feels that he has a heart. When this takes place the symptoms do not differ from those of cardiac failure brought about from valvular disease.

The physical signs of an hypertrophied left heart are increased tension in the radials, well-marked apex beat and accentuated aortic second sound. When the right heart is hypertrophied we have an accentuated second pulmonary sound. These signs are only of value as pointing to increased tension in the various vessels, and when this has lasted some time we naturally conclude that there is hypertrophy. If, however, a cause which has been in operation for some time is no longer present, the signs of increased tension will have disappeared, although the hypertrophy remains. Percussion is no doubt of value in many cases in detecting marked hypertrophy, but for slight degrees it can give us no reliable information. The shape of the chest has much to do with the extent of cardiac dulness. When it is barrel-shaped, a large heart will present a no greater degree of dulness than a small heart in a flattened chest. A great degree of dulness can only take place in dilatation. Again, a large heart may be more or less entirely covered by an emphysematous or hypertrophied lung.

Dr. ADAMI discussed the anatomy and experimental pathology as follows:

I shall, I think, best satisfy you, and at the same time myself, if what I contribute to this evening's discussion takes the form of a series of notes upon the experimental pathology and the anatomy of cardiac hypertrophy, rather than that of an academic survey of the subject from the clinical standpoint. Frequently, it is true, I must of necessity illustrate what I have to say by reference to clinical history, but, on the whole, I shall leave the clinical aspects to be dealt with by those more capable.

In the first place, if we study the causes of hypertrophied heart, whether of hypertrophy of one or both sides, we see this that reading the clinical history of these cases the assigned causes of hypertrophy may be summed up under the heading of *increased work*. This one heading may be subdivided into three,—increased work due to resistance from within, increased work due to resistance from without, increased work due to nervous stimulation and augmentor action. I shall not discuss this last subdivision, because frankly we are ignorant how far the hypertrophy that occurs in exophthalmic goitre and allied conditions is due to heightened blood pressure, and how far it is secondary to excitation of the accelerators or augmentors.

Of the increased resistance from within, or increased tension, the main causes are, heightened pressure in the arterial blood stream, and secondly, obstruction to the onward passage of blood within the heart itself, by stenotic diseases of one or other orifice. Of resistance from without, the one great cause is pericardial adhesion. To-night we have, as far as possible, to leave out the subject of valvular disturbance, and I shall neglect nervous disturbances. There is still the large field of hypertrophy due to increased arterial pressure, and the pericardial adhesion. In all these cases, the individual fibres of the heart muscles of the affected regions have to contract under increased difficulty, they have to carry or contract against a greater load, and as a result of this, just as is the case with the skeletal muscles, with the muscles in the blacksmith's arm, and the muscles of the body in the all-round athlete, increased work brings about increased growth—brings about, that is to say, hypertrophy of the muscle.

Into the subject of the nature of this increased growth I shall enter in a few minutes' time, at present I wish to carry a word further this parrallel between the behavior of the cardiac and skeletal muscles, under circumstances in which the load is increased. If you take a skeletal muscle, for example, the gastrocnemius of the frog, so dear to the physiologist, and observe its contraction with gradually increasing loads, there are two points especially to be made out. In the first place, the greatest amount of work is not performed with the smallest load, but there is a certain medium load with which the distance through which the load is pulled multiplied by the weight of the load gives the biggest result. This product of weight moved and the distance through which it is moved is the work done by the muscle. The most work, therefore, is done with a medium load. The second point is that with increasing weights fastened or brought to bear upon the muscle, that muscle in its resting state becomes more and more elongated, and with regularly increasing weights attached, the shortening attained by the contracted muscle constantly diminishes. Or, to put the matter in a slightly different light, and to combine these two statements of fact, although with a certain medium load the greatest amount of work is done, nevertheless with that medium load the muscle in contracting does not attain to the same amount of shortening as it does with a lesser load.

Let me now apply these observations to what