is found in the ventricular muscles of mammals. Experimentally, the amount of work performed by the ventricles of the mammalian heart can be increased by ligaturing the aorta with a sip-loop ligature, and drawing this ligature more or less tight, according to need. (This is an animal that has been narcotized and curarized and subjected to artificial respiration, the heart being exposed by making a window in the ribs.) In such a case as this, as shown by Professor Roy and me,* the behavior of the cardiac muscle can be observed and recorded by an apparatus, of which I give a rough diagram. The ends of this apparatus are attached to the surface, say of the left ventricle, by fine threads, and now it is possible to observe upon the recording drum the extent of contraction of the portion of muscle between the two points under different pressures within the heart. Narrow the aorta by drawing the ligature tight and the pressure is increased. Under these conditions it is found that the ventricular muscle reacts exactly along the same lines as does the gastrocnemius of the frog.

Similar results are obtainable if, instead of increasing the pressure in the arterial system by narrowing the aorta, we increase the work of the heart by increasing the amount of blood passing through it, either temporarily, by pressure upon the abdomen, whereby a large quantity of blood is expelled from the abdominal viscera, or by injecting into venous circulation some few hundred cubic centimetres of defibrinated blood. The results in all these cases are the same. By the instrument just described it is easy to see that the heart is more filled in diastole, so that the two ends of the levers are pushed further apart, and that in systole the ends do not approximate so nearly as in the condition when there is less resistance or less blood pouring through the organ.

It is seen from these observations that with increased pressure with the ventricle the wall expands in diastole. There is dilatation of the heart. But with the increased load to contract against, the fibres do not shorten to the same extent;—that is to say, with increased work of the heart there is, necessarily, accompanying the dilatation in diastole, a dilatation in systole. All the blood is not expelled in systole. There is of necessity *residual blood*, as Roy and I termed it, in the ventricular chambers.

There is a general belief that the healthy heart, even under conditions of increased work, contracts completely, so that the chamber is emptied at the end of systole. From what I have said it will be seen that this is not the case. One can go further and prove for one's self that even under ordinary conditions the mammalian heart does not completely expel all the blood within the ventricles. By taking a dog that has been curarized and subjected to artificial respiration, opening the chest wall, making an incision at the very apex of the left ventricle, so as just not to completely enter the cavity, then it is easy to push the little finger into the cavity through the thin apex without the loss of a drop of blood. The heart action is not recognizably disturbed by this procedure, and it can be felt that while the walls of the ventricle in the lower two-thirds up to the aspices of the papillary muscles close completely round the finger, there is clear space in the upper third which is not and cannot be emptied of blood.

Although it may seem at first sight to have no direct bearing upon the subject of this evening's discussion, nevertheless it is worth while to make a few remarks upon this subject, inasmuch as it is so intimately associated with conditions of hypertrophy without valvular disease. It is quite possible that where there is increased work to be performed by the heart, there is some economy of the action of the organ when there exists a certain amount of residual blood in and dilatation of the ventricles. Taking the ventricular chamber as a sphere,* there is this to be noted concerning the relationship between the circumference of the sphere and its contents, namely, that as a sphere expands, its cubic contents increase out of all proportion, I was going to say, to increase in cubic contents and increase in circumference is by no means an arithmetic ratio.

If the circumferences be taken as abscissæ, and the corresponding volumes as the ordinates, the curve of successive values is what is known to mathematicians as a cubical parabola. From this it follows that a degree of shortening of the fibres of the heart wall, sufficient, let us say, to reduce the circumference of the ventricle one inch, will cause a greater diminution in *volume* (a greater output) the more dilated the ventricle is at the beginning of its contraction. For example, a diminution of the circumference by *one* inch of a sphere whose circumference is *ten* inches causes a diminution of the volume or an output, in the case of the heart, equal to 4.5 cubic inches, while a diminution by *one* inch in the circumference of a sphere *five* inches round causes a diminution or an output of only 1.027 cubic inches, although in the first case the circumference was reduced only by one-tenth, while in the other case it was reduced to one-fifth. That is to say, if we have a dilated heart, the fibres will need to contract a very small amount in order to expel a given amount of blood, compared with the amount they would have to contract in the normal undilated heart.

There are other factors to be taken into account, it is true, and Roy and I went a little into this subject in our paper published in the

* Heart beat and pulse waves, *Practitioner*, February, 1894, p. 81.

* The sphere is the nearest geometrical figure that can be employed here for purposes of illustration.