aortic regurgitation are worthy of careful consideration, and teach us a most instructive lesson in the study of cardiac pathology. This hypertrophy of the heart is the means by which dilatation is arrested rather than a measure to increase the power of the ventricles, and enable it to overcome some obstruction. We are much too apt to assume that the latter is essentially the condition which evokes hypertrophy. I will arrange the evidence as briefly and tersely as the subject permits. 1. Hypertrophy does not always follow an obstruction to the flow forward of the blood on the ventricular systole. Though it is the common result, it does not always form the result. In anæmic systems, in chronic Bright's disease, dilatation is found instead of hypertrophy; this is especially seen in women. 2. Hypertrophy is found under circumstances where there is no obstruction to the blood flow, notably in the hypertrophy of the left ventricle so commonly seen in mitral regurgitation (Niemeyer, vol. i, p. 318, ed. of 1870). Again, as the same authority tells us, hypertrophy is frequent in cardiac dilatation, the result of partial myocarditis accompanying pericarditis (p. 298 of the same edition), by which the dilatation is limited. In both these instances, there is no obstruction to overcome, but in each there is a dilating process to be arrested. In mitral regurgitation, the blood rushes into the left ventricle with unwonted force from the distended auricle and veins behind it, and a dilating process is so set up, which, in well nourished organisms, is limited by a growth of muscular fibre. There is no similar enlargement of the left ventricle in mitral stenosis, though there the auricle and pulmonary veins are equally distended; but then there is an abnormally small, and not an unnaturally large orifice, through which the blood can flow furiously into the left ventricle. In the case of softened walls, leading to dilatation by the normal inrush of blood, the dilating process sets up hypertrophy sooner or later; sooner in well nourished organisms, later in debilitated systems. How dilatation induces hypertrophy, cannot be given here. It is not the place, even if the time could be spared.

This brief digression will enable us all the more readily to see that, in pure aortic regurgitation, the growth of the walls of the heart is not the result of any obstruction to be overcome; for none such exists, or, if so, only to a trifling extent. But there is a dilating process to be arrested. When the aortic valves are rendered incompetent, the left ventricle is no longer filled solely by the blood coming in from the auricle and pulmonary veins—a comparatively calm current—it is also filled by a second blood-current—the blood driven backwards by the aortic rebound through the insufficient aortic valves. The distending power of this new current is a very different matter from the normal current, which is itself undiminished. There is, indeed, no diminution in the normal distending force, while there is added to it a new force of unusual and unwonted power. The ventricle is now, in fact, distended by the aortic recoil, and the regurgitant current possesses great distending force. The ventricle yields before this new force, and dilatation would soon become marked and the ventricle be placed *hors de combat*, if it were not for the hypertrophy which, coming to the rescue, arrests the dilating process and limits the dilatation. It is under these circumstances, indeed, that we find the most massive hypertrophy—the *cor bovinum* in fact. The hypertrophy is not to overcome obstruction here, but to arrest dilatation. It is necessary to be clear about this, in order to comprehend the indications for treatment. We do not, under these circumstances, require more forcible ventricular contraction—the effects of digitalis; for the powerful and enlarged ventricle is already working ruin in the arterial walls, which, at every systole, are distended by the contraction of a ventricle, not only much more powerful than a normal ventricle, but holding a larger quantity of blood. The overdistension to which the arteries are subjected produces chronic parenchymatous inflammation of their walls, or atheroma (see an article by the writer in the *Philadelphia Medical Times*, August 7th, 1875, on "Atheroma"), and to administer digitalis here is to aggravate the evil. Already the contraction of the enlarged ventricle overdistends the arteries, so that the pulsation may be seen in the arteria centralis retinæ with the ophthalmoscope; and, to administer an agent like digitalis, which, as Balthazar