emunctories and lessening the amount of fluid in the body. Baths play an important part in this latter respect. A recent writer who studied the action of baths at Nauheim, Germany, in vavular disease, known as Schott's disease, believed they diminished the size of the heart by peripheral dilatation of the vessels, besides increasing the sink transudation. Brine baths, carbonic acid baths, and hot baths were all employed for this effect. The patient came out with a skin as red as a lobster, and the area of hypertrophy and dilatation as determined by percussion was sometimes reduced as much as one inch as the result of a single bath.

Dr. FINLEY thought too much stress might sometimes be laid upon arterial sclerosis as a causative factor in the production of cardiac hypertrophy; that it might sometimes be a secondary rather than a primary condition, although, with the sclerosis once established, a vicious cycle was set up, in which enlargement of the heart and sclerosis produced and kept up each other. He believed, however, that some cases occurred in which the cardiac trouble was the primary one. Cases of aortic regurgitation were not uncommon in young people where a considerable degree of arterial sclerosis existed. In Graves' disease Dr. Adami seemed to think that the enlargement of the heart was due to increased arterial tension; now, in these cases the arterial tension was low. It seemed to Dr. Finley that the cardiac changes that occurred in Graves disease might be the result of the increased work thrown on the heart by the increased number of pulsations. As to the symptoms of cardiac hypertrophy, one may say there were none. It was when dilatation supervened or when compensation was defective that symptoms occurred. They were, of course, similar to the symptoms following mitral disease when compensation was failing. All kinds of pulse were met with in this condition; sometimes weak and irregular like the advanced stage of mitral stenosis; sometimes in pairs, one weak and one strong; and the few cases of brachycardia and tachycardia seen by Dr. Finley were associated with this condition. He regretted that physiology had not been able to do more to clear up this subject; so far, experimental work had thrown very little light on the irregularity of the pulse. Touching the treatment, he believed it a good rule to divide the cases into two classes: (1) those of high tension, (2) those of low tension. In the first the object should be to decrease tension, and iodide of potassium was often very useful in relieving distressful attacks of palpitation; nitro-glycerine had its uses, and purgatives, especially mercurial, followed by a saline in the morning, were of very considerable value. For the purpose of relieving sleep and distressing dyspnœa, nothing equalled morphia. The other hypnotics, such as sulphonal, chloral, paraldehyde, often failed. In the second class attention should be directed towards strengthening the heart and giving the ordinary cardiac tonics.

Dr. LAFLEUR, seeing that Dr. Stewart had exhausted the etiology of the subject, would content himself with reading a tabular statement of cardiac hypertrophy in general. It was based as follows: (1) Causes within the heart: these were practically two: (*a*) myocarditis, however induced, either sclerotic, or that which is the result of chronic pericardial inflammation; (*b*) aneurisms, which by weakening one portion produced hypertrophy in others. (2) The second great division included causes outside the heart; among these were noted: (*a*) purely mechanical causes, and of these the principal and only one was in reality adhesion of the pericardium, synechia pericardii, which might or might not be combined with pleural adhesion; (*b*) a great number of causes which depended upon the raising of blood pressure. Here the distinction might be made of blood pressure raised in territorial areas, or a general increase of blood pressure; among the former were chronic or subacute nephritis, chronic pulmonary diseases, chronic bronchitis, sclerosis of the lung, and true chronic fibroid phthisis. Still dealing with territorial raising of blood pressure, we had the pressure of tumors upon large vascular trunks (quite a rare cause, but it might occur in mediastinal disease). Then the general raising of blood pressure, as brought about by poisons of various kinds; by excessive manual labor; nervous derangements; and arterial sclerosis. (3) Hæmic plethora. This was not infrequently combined with arterial hypoplasia. Dr. Lafleur remarked he had access to some statistics which showed the proportion in which these various causes come into effect, drawn from 360 autopsies representing the total number of autopsies from the opening of the Johns Hopkins Hospital, May, 1889, to April, 1893. In 360 autopsies, cardiac hypertrophy, due to some cause or other, was found to exist in no less than 105 cases. Of these, arterial sclerosis was found to be the cause in 59%; chronic nephritis in 13.4%; valvular lesions, 12.4%; adhesions of the pericardium in 7.6%; excessive muscular work in 3.8%; tumors, 1.9%; aneurisms in 0.95%; hæmic plethora in 0.95%. It was seen from this paper that more than 50% of the cases of cardiac hypertrophy in general hospital work was due to arterial disease. The frequency therefore of arterial sclerosis had certainly been underestimated. The speaker knew it to be extremely common in the United States, and, from all reports, it was so upon the continent of Europe. He agreed with Dr. Adami that dilatation, in the vast majority of cases, accompanied hypertrophy. Concentric hypertrophy was almost always a post-mortem change. An observer (Corvisart), during the