

tive to motion, the cycling acting upon it like a stimulant. The over-development of the heart under the continued and extreme over-action affects in turn the resilience, modifies the natural blood-pressure, and favors degenerative structural changes in the organs of the body generally. Every medical man here must, I think, have met with instances of cardiac hypertrophy in athletes. There can be no doubt, that in such a violent game as lacrosse this condition develops. Cardiac hypertrophy from overwork may be recovered from, or it may go on to progressive heart failure. It develops more rapidly, and more often affects those who at the same time use alcohol in any form to excess.

It is easy to understand how severe muscular efforts bring about hypertrophy of both ventricles.

Very rare causes of cardiac hypertrophy are congenital narrowing of the arteries and general dilatation of the blood vessels. The former is, according to some, far from rare, and it is held that in many cases of enlargement of the heart from over-exertion the predisposing cause is congenitally narrow vessels.

In severe cases of lead poisoning cardiac hypertrophy is very commonly met with, due mainly to the parenchymatous and interstitial changes in the kidneys and arterial sclerosis. These arterial and nephritic changes are very constant phenomena of severe lead poisoning.

The explanation usually given of the action of lead on the kidneys and arteries is that it induces gout. Sir William Roberts, however, believes that lead does not induce gout. He considers that the gouty diathesis and lead poisoning, while differing in all other respects, have one tendency or vice in common, viz., the tendency to uratisis. However the facts may be explained, there can be no doubt about the influence of lead in bringing about sclerotic changes in the kidneys and arteries, and thus leading to hypertrophy. These changes, next to the encephopathy, constitute the most serious effects of lead poisoning, effects which, if their cause is not early recognized, will infallibly lead to irretrievable mischief.

Cardiac hypertrophy is the most constant change in the heart in gouty subjects. It is nearly always present, differing in degree according to its intensity and age of the patient. It is in some cases combined with dilatation and myocardial changes, especially fatty degeneration. It is caused by the widespread arterial and kidney changes so common in gouty subjects. In forty-nine cases examined by Dr. Norman Moore, the average weight of the heart was  $16\frac{1}{2}$  ounces. Gout may cause hypertrophy without first bringing about arterial organic changes. In this connection it will be convenient to discuss the cardiac hypertrophy which is so frequently found in cases of sub-acute and

chronic Bright's disease. The variety of Bright's disease which is most frequently attended by cardiac hypertrophy is the interstitial. For many years the connection between the circulating and kidney changes has been a subject which has given rise to a great deal of discussion.

There is no doubt that cardiac hypertrophy may occur in a simple Bright's disease without any involvement of the general arteries. We have probably two factors contributing to the hypertrophy, the increased pressure in the circulation caused by the necessity of getting rid of waste matters. As a great number of capillary districts are obliterated by the disease, the heart must increase in strength in order to effect the necessary elimination; but the chief cause for the hypertrophy of the left ventricle is the retention in the blood of matters which in a normal state of the kidneys would be eliminated. This causes high arterial tension and gives rise in consequence to increased work and consequently to hypertrophy.

The last group of causes giving rise to cardiac hypertrophy which it is my intention to speak of is the neurotic group. To this belongs the enlargement coming in exophthalmic goitre, essential tachycardia, insanity, prolonged emotional disturbance, etc. The excessive action of the heart in these cases leads to enlargement, but little is known about the intimate changes in the nervous system which brings them about. Our knowledge of the changes which take place in the nervous mechanism of the heart is very slight indeed. It is highly probable that in the case of heart changes coming on during the course of exophthalmic goitre, essential tachycardia and from the excessive use of tobacco, tea and coffee, the changes are of a molecular nature. There are very good grounds for believing that all the essential symptoms of exophthalmic goitre are due to the action of certain toxins generated from the thyroid gland. Possibly it may be found that essential tachycardia is brought about also by a chemical poison generated within the body. The hypertrophy and dilatation of the heart occurring in exophthalmic goitre reaches sometimes an extreme degree, and cases are not uncommon where death is the direct result of degeneration occurring in the heart muscle. I am not aware of any fatal case of tobacco-heart. It is quite possible that such a heart might give way at last under the continuous strain, but fortunately the cause is easily detected, and if removed the effects disappear, although to this there are rare exceptions. An abiding palpitation has been described even after tobacco has been given up.

The enlargement of the heart occurring in the course of infectious fevers is mostly due to dilatation of the cavities, and hence is properly beyond the range of my subject. The subject