

say that while one can, in certain cases of hypertrophy, make out clearly that the individual fibres have undergone a definite increase in size, it is far more common to note, and of this there can be no doubt, that there has been an actual numerical increase in the fibres. This increase appears to be general throughout the ventricular wall, and is possibly, nay probably, due not only to a new growth beneath the endocardium especially, but also to a splitting up or division of pre-existing fibres. It must be remembered that the heart muscle fibre is not a single cell, but is a compound, the result of a fusion of several cells into one individual unit. As a consequence of this it is possibly more easy for the fibres to split up into independent territories without undergoing temporary derangement of function than is the case with the cells of those tissues formed of isolated cell units.

To pass on now to certain aspects of this subject of hypertrophy more immediately in connection with this evening's discussion, I would point out that of the cases of hypertrophy without valvular lesion, we have to consider in the first place increased resistance through the column of blood. This could be brought about by increased amount of blood to be propelled, or, in the second, by increased resistance to passage in the arterial system. Of these two the first may exist as a constitutional condition, but the more one studies the less assured does one become that there is such a condition as general plethora unless these cases be regarded as true plethora in which (as in German beer drinkers) there is oft repeated flushing of the circulation with imbibed fluid. Of increased resistance in the arterial stream the reverse would appear to be the case, and with further studies of blood pressure in the arteries one begins to see that this plays an extremely important part. The hypertrophy following upon not only gouty conditions and senile arterio-sclerosis, but also upon acute rheumatism, chorea and chlorosis, may be present with or without lesion of the aortic or mitral valves of sufficient intensity to explain its extent; so that in all these cases we have to fall back upon increased blood pressure as a cause of hypertrophy.

Increased blood pressure in itself is capable of setting up a vicious circle of which one segment may be hypertrophy.

In the first place it leads to an increased nutrition of the walls of the arteries, increased nutrition leads to increased connective tissue growth of the walls, the increased fibrous tissue of the walls leads to contraction and increased rigidity of those walls, the increased rigidity leads to increased resistance to the passage of the blood current, the increased resistance required increased propulsive power on the part of the ventricular

muscle, that is to say, increased work; the increased work of the heart leads to overgrowth and hypertrophy, and with this, heightened blood pressure and further increased nutrition of the walls. And now at last the stage is reached, this vicious circle continuing, in which either the walls give way or the heart.

The longer I study the pathology of the circulation—and during the last eight years I have given more time and thought to this than to any other branch of my subject—the more assured do I feel that increased blood pressure alone (however it be primarily brought about) is sufficient to explain the anatomical changes so constantly seen in arteries, valves and heart walls, without of necessity calling in chronic inflammation or specific agency. The changes I refer to are arterio-sclerosis, atheroma, and general fibroid thickening of the valves. Perhaps here again I am diverging from the main subject of this evening's discussion, but I say this as a connecting link with what I have just remarked and with what is about to follow.

While I am far from wishing to indicate that this is to be regarded as the sole cause of atheromatous and arterio-sclerotic changes, I hold that the changes I have mentioned can one and all be explained by the increased pressure within the vessels leading to an increased passage of fluid from the blood into the sub-endothelial layers of the intima, to an increased nutrition, and as a consequence to a proliferation of connective tissue in this region, which in itself as it contracts cuts off its own supply of nutrition, degenerates, and, what is more, leads to degeneration of surrounding parts by cutting off their nutrition. The evil effects in arterio-sclerosis, with all its combined lesions, are not necessarily of an inflammatory origin.

Let us take now the hypertrophied heart. Time permits me to refer but briefly to the anatomical changes that may occur in it in the cases before us.

1. The overgrowth of the arterial walls may be associated with an increased tendency to the development of fibrous tissue in the immediate neighborhood of the arteries, and thus a condition of so-called interstitial myocarditis may be set up; or

2. With an increased fibrosis of the arteries the narrowing of the channel may lead to incomplete nutrition of the territory supplied by each arterial twig, and as a consequence the muscle fibres at the periphery of the territory may be atrophied through lack of nutrition and be replaced by fibrous tissue. This is the so-called dystrophic sclerosis of the French school, and can frequently be seen more especially in the papillary muscles.

3. With the arterial disturbance there may