The type of dropsy in these animals, therefore, corresponds more closely to that of heart disease than to that seen in disease of the kidney or in hepatic cirrhosis, in both of which conditions the oedema fluid contains much less solid matter.

This conclusion is not at variance with the pathology of cardiac ascites, because, as shown by one of us,¹ in that condition there may be no changes of pressure in the peripheral circulation.

REFERENCE.

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THE HISTOLOGY OF RHEUMATIC CARDITIS AND OTHER RHEUMATIC PHENOMENA.

BY

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Heart.—In all three parts of the cardiac wall certain inflammatory lesions, which have been called "submiliary nodules," are found. These nodules consist of (a) a groundwork of homogeneous material which gives the staining reactions of fibrin; (b) a more or less defined zone of cellular proliferation, the characteristic cells being large and of a fibroblastic type, with a deeply staining cytoplasm, and from one to six nuclei in which a somewhat loose chromatin skein takes haematoxylin with avidity. They are therefore similar in structure to the larger subcutaneous node so characteristic of rheumatic infection. The shape of the nodule is dependent to some extent on its surroundings, being globular when lying in a loose bed of connective tissue, but fusiform where it is limited laterally, as it is when it develops in the interstitial tissues of the myocardium. In rheumatic lesions of the heart degenerative and necrotic changes are not intense in degree; exudation of leucocytes is moderate and diffuse, mononuclear types predominating.

Myocardium.—Recent work has confirmed the conclusions already stated.¹ The nodules are closely related to coronary arterioles; there are more in the wall of the left than in that of the right ventricle (though in one case the only nodule found was in the wall of the right ventricle) and only once has a nodule been found in the auricular walls apart from a few in contact with the mitral ring. In this case it lay near the base of the Eustachian valve in the interauricular septum. In two cases a nodule has been noted in the connective tissue limiting the *a-v* bundle, and in another case arterial changes in this region coincided with nodule formation at the point of junction between the *a-v* node and the bundle. In one case segmentation has been observed, and in another a close relation between nodule formation and the distribution of intracellular fatty change was seen. On the whole, the muscle cells appear less altered than might be expected, probably owing to the relative coarseness of the staining methods at our disposal.

Endocardium.—Here again recent investigation has merely confirmed observations already recorded.² The points of main interest are the almost constant occurrence of nodules in the deeper parts of the valves, the presence of a diffuse and mild mononuclear leucocytosis, and the ultimate vascularization of the inflamed valve. The endothelium proliferates; the surface cells float off owing to degenerative changes, and the fibrin deposited in this stripped surface—sometimes mingled with leucocytes from the intracardiac blood—becomes permeated with new connective tissue bearing young capillaries.

Pericardium.—The chief facts are that nodules have been found in all cases of active pericarditis, but not in quiescent cases. They have not so far been seen in the parietal but only in the visceral layer, where they are scattered over the cardiac surface. They lie at a varying distance beneath the endothelium, in which proliferation,

superficial degeneration and stripping, fibrin deposit and capillary formation, are seen as in the endocardial endothelium. In the subendothelial tissue a moderate leucocytosis is seen.

The *aorta* has been examined several times but without the discovery of such lesions as have just been described. Mesenteritis and small patches of subendothelial inflammation have been seen, but there has been nothing in these to distinguish them from the similar lesions which occur in most infections.

Arteries, Arterioles, and Capillaries.—In one case the lumen of a coronary arteriole in the vicinity of the auriculo-ventricular bundle was almost closed by swelling of the arterial wall on one side, due apparently to the development of a nodule in its middle coat. Endothelial proliferation with thrombosis has been noted also in the arterioles and capillaries of a rheumatic joint. Coronary periarteritis was alluded to in the paper on myocarditis.

The central nervous system has been more or less fully examined in four cases of chorea.

Here it may be said that widespread chromatolysis is present throughout the cortex, and to a less degree in the optic thalamus and the pontine and medullary ganglia, while in the cord almost no changes were discovered. The pia mater, which has been fully examined both on the convexity and at the base, shows no change beyond intracapillary clotting and an occasional small extravasation of leucocytes. The one is probably due to *post-mortem* clotting and the other to passive hyperaemia.

Only two joints have been available. In one, a large and very definite nodule was found, with others less clearly defined, corresponding essentially with those seen in carditis. It lay just beneath the endothelium, which at other spots was proliferating. Throughout the synovial membrane and extending into the periarticular tissues was a mononuclear leucocytosis; there were further, the vascular changes already noted.

The other organs examined have been the lungs, in which an intense passive hyperaemia was seen, together with a mild bronchial catarrh; the lymphatic glands at the base of the heart, in which passive oedema and congestion were manifest; the spleen and liver; the kidney, in which spots of parenchymatous degeneration have several times been seen; the tonsils, thymus, and adrenals; and the thyroid gland from a case of Graves's disease with chorea, in which the changes peculiar to the former disorder were found. In none of these organs has any kind of interstitial inflammation been seen.

Other types of cardiac infection have been examined by way of comparison with rheumatic carditis. In practically all cases of destructive endocarditis and purulent pericarditis foci of inflammation are found in the myocardium; but in no such case has an inflammation of all three layers been seen. Further, the inflammatory reaction is chiefly characterized by an exudation of leucocytes, usually polymorphonuclear, not by a proliferation of the cells of the part invaded. In one case of protracted cardiac and articular rheumatism, terminated by a lobar pneumonia from which the pneumococcus was readily isolated, two sets of lesions were visible side by side in the heart, and even in the same section—the one characteristic of rheumatic infection, the other a polymorphonuclear focus. In one of these latter pneumococci were demonstrated.

Finally, it may be added that examination of lesions produced by inoculation of animals with micro-organisms said to be the cause or causes of rheumatism is proceeding. A good deal of material has been dealt with, but any statement as to the results would be premature at this stage.

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¹ *Quarterly Journal of Medicine*, October, 1908. ² *Lancet*, May 15th, 1909.