

was somewhat relieved of cholemia. There was a free discharge of very dark colored bile. The autopsy revealed a large malignant nodule in the transverse fissure, which completely obstructed the branches of the hepatic duct. The biliary canals throughout the liver were very much distended.

In July and August of this year, 1898, I conducted a number of experiments on dogs to determine the value of incisions in the liver in relieving biliary obstruction.⁵ Five dogs were killed and the liver injected with fluid either through the gall-bladder, the common duct having been tied, or through the common duct. Incisions were then made in all parts of the liver, with the result that streams of the fluid would issue from the bile canals and there was general oozing of the fluid from the wounded surfaces of the organ if much force were used. Six dogs were anesthetized and the liver injected as above and this fluid with blood would flow as a rule freely when much force was used in injecting the fluid. Four had the common duct tied and after twenty-four hours the same experiment was conducted under anesthesia with similar results. In nine the common duct was ligated and gauze packed around the field of the gall-bladder and ducts. After from twenty-four hours to a week the liver would be incised in one or more places and as a rule the bile escaped satisfactorily through the gauze. It would be very dark after prolonged obstruction. Two of the dogs died, and the others were killed in from five days to two weeks after the second operation. Before killing them and while under the anesthetic, fluid was injected through the bladder or duct, and would flow from the wounds which had been made in the liver at the second operation, and also from incisions made at that time. Five had the gall-bladder removed and cystic and common ducts ligated, but as gauze was not used at the time of the operation to wall off the general cavity, they died in from twenty-four to forty-eight hours from shock and from the escape of bile from the liver where gall-bladder had been removed—the obstruction in the duct forcing bile from the liver.

Other experiments were conducted, but they added nothing to the above. While, as I have stated, the field for hepatotomy in gallstone diseases is very restricted, yet in those few cases it offers the surgeon much assistance in bridging the patient over till in better condition for a radical operation.

As we advance in the surgery of the ducts the field for cholecystenterostomy grows smaller. Certainly it should not be done, leaving a stone in the duct. Malignant disease and agglutination of the duct call for it. The Murphy button makes the operation quite simple.

Treatment of Hypertrophic Rhinitis with Zinc Chlorid.

—A few drops of a 10 per cent. solution of zinc chlorid injected under the mucous membrane, is an effective means of curing hypertrophic rhinitis in those cases in which the swollen mucous membrane is smooth, soft and easily depressible, and by its distention over the lower turbinates obstructs the nasal passage. When it is thick and resistant this method has no chance of success. Viollet reports in the *Gaz. Hebdomadaire* of August 18, that he has thus cured 5 out of 8 patients, the improvement unaltered for more than a year to date. His experience is much more extensive, but he omits those not scrupulously traced. His failures were due to his ignorance at first of the conditions of success as above described.

5. Experiments were conducted at the Birmingham Medical College, and I was assisted by Dr. R. E. Hogan, Assistant Professor of Gynecology and Abdominal Surgery.

Original Articles.

THE ROLE OF THE MYOCARDIUM IN PERICARDITIS.*

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Pericarditis is a condition of danger in any one or all of the following ways: 1, as a focus of infection; 2, as a cause of mechanical or reflex disturbance of the heart action; 3, as a starting point for a spreading inflammatory lesion.

As a focus of infection its danger is greater than is that of pleurisy or possibly even of peritonitis. The exudations of the pericardium are habitually more hemorrhagic or purulent than are those of the pleura in similar or the same infection and the clinical indications of general infection are also significant of a more virulent process.

The mechanical dangers are usually of little consequence, and, as has been found in the case of endocarditis, wider knowledge gives evidence that the circulatory disturbances of pericarditis are occasioned by changes in the heart muscle to a much greater extent than by mechanical or reflex disorders of the organ. The association of endocarditis with pericarditis is especially common in childhood, and either process may be the primary or they may occur simultaneously. The resulting condition is generally a heart so crippled that the continuance of life beyond the time of adolescence is purchased at the price of the extremest watchfulness. Fortunately, this association is not a very common one.

I hasten to explain that my reference to the two conditions, endocarditis and pericarditis, in such a way as to indicate that they constitute a pathologic entity, is largely out of deference to usage now somewhat antiquated. There are cases that we may on this ground term endo-pericarditis, but the essence of the malady is a "pancarditis" and its dangers are proportioned to the completeness with which the whole of the heart is involved. There are cases of pericarditis with very slight involvement of the underlying muscle, cases with considerable muscular disease and slight endocarditis, and finally, instances of severe involvement of all three. The last are the cases that have customarily received the name endo-pericarditis.

The myocardial element has not been unrecognized by the pathologists, but clinicians are only beginning to realize its significance. Chronic myocardial disease as a cause of clinical symptoms does not constitute a new conception. The degenerations have long been looked upon as important clinical conditions; of these, curiously enough, fatty degeneration has until recently fairly monopolized the clinician's attention, though the far more important condition is fibroid degeneration or inflammation. This may be independent of any other cardiac disease or it may be associated with endocarditis or pericarditis. The independent form is of greatest interest at the present time because its development is less clearly known, and its existence is less certainly recognizable. This independent myocardial fibrosis, or myofibrosis cordis, however, is not a matter of special interest in our discussion.

* Read in a Symposium on Pericarditis at the Fifty-second Annual Meeting of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee of the Section.

Of more importance is the fibrosis accompanying chronic pericarditis or endocarditis. In these cases the myocardium becomes affected by direct extension or in consequence of circulatory disturbances. The terminal result of this condition, as in independent myofibrosis, must always be cardiac dilatation, if intercurrent disease have not prematurely terminated life. Before, however, this terminal condition has developed, clinical signs of failing circulation may arise as obscure indications of the myocardial weakness associated with the pericardial disease.

I shall not allude to the physical signs of simple pericardial disease, such as frictions, mammary or costal retraction, etc., but would recall the symptomatic and physical evidences of muscular incompetency in cases of combined pericardial and muscular disease. As in the independent myofibrosis, there may be a gradual loss of vigor, a premature senility, a tendency to irregularity of the heart to a peculiar grayish pallor, to gastric disturbances, etc., but all of these indications are less prominent than in the independent form because chronic pericarditis is more frequent at an early age, when the senile type of clinical manifestations are unlikely to occur.

The best evidences of myocardial association with pericarditis are therefore those derived from physical examination. First of all I wish to insist that marked hypertrophy of the heart develops in a surprisingly short time in acute cases. In a recent case in a boy of 9, I found such rapid enlargement of the heart that it seemed impossible that the enlargement could be other than dilatation. The autopsy, however, showed practically no dilatation and very great thickening of the walls. This enlargement, however, is not true hypertrophy; it is largely degenerative, and the clinical signs would indicate this. The pulse is weak and of low tension—out of all proportion to the degree of enlargement of the heart. The slapping, irritable impulse of Martius accompanied with a weakened and quick pulse is of prime importance in indicating myocardial association with the pericardial disease.

What I have just said refers especially to acute cases. The chronic cases are attended with less pronounced signs of myocardial disease, but it may be said with considerable emphasis that peripheral congestion or cyanosis, the peculiar enlargement of the liver that has been termed pericardial cirrhosis of the liver, the occurrence of dropsies and marked irregularities of the heart, are always indicative of associated myocardial trouble and therefore of grave prognostic significance.

I do not wish to be understood as saying that myocardial disease always accompanies pericarditis. In a strict pathologic sense there is probably always some involvement of the muscle of the heart, at least to the extent of slight sub-pericardial infiltration, but in a clinical sense the heart wall is often unaffected. It should, however, be recognized that even such slight myocardial disease may occasion striking signs and symptoms. Thus, Fisher has found great disturbance of the heart action and of the sounds—a Flint's murmur—when there was only a moderate sub-pericardial disease. In this connection I wish also to recall that Romberg has found in simple endocarditis thrombosis of the myocardial vessels and myocardial degeneration. This contribution is of very great importance, for in no other way could the marked disturbances of the heart's action be explained in cases of endocarditis with anatomically trivial lesions. I would couple this with what occurs

in pericarditis. The symptoms of the latter are largely the result of the underlying myocardial disease.

We are thus forced to the conclusion of Jürgensen that pancarditis is the diagnosis of the future, its type may be endocarditic, pericarditic, or myocarditic, but the immediate result and the final prognosis are to a large extent dependent upon the degree of involvement of the myocardium. A few dangers, such as general infection, embolism or mechanical interference with the heart by extensive effusion, are independent of the condition of the heart wall, but, these conditions excepted, the important criterion for prognosis is the condition of heart muscle.

ADHERENT PERICARDIUM.*

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It is so manifestly impossible in the few minutes allotted to me to consider this subject in its entirety, and so many of the points bearing on this subject have already been considered by preceding speakers, that it seems best to restrict my remarks to the clinical aspects of this disease.

We meet with adherent pericardium in two forms: 1. as a result of pericarditis interna which has led to a more or less complete and firm union of the two layers of the sac, without adhesion to the surrounding structures; 2, as a result of pericarditis interna et externa which has caused adhesion not only between the pericardium and epicardium, but also between the sac and the contiguous structures, as the chest wall, diaphragm and lungs.

In this second form there is often such an extensive development of fibrous tissue within the mediastinum, with consequent union of all the structures therein contained, that the condition has been termed chronic adhesive or fibrous mediastino-pericarditis. In some cases this proliferation of fibrous tissue is not limited to the mediastinum, but invades the pleural and peritoneal cavities in the form of a general serositis.

Not only is the capsule of the liver thickened, but the connective tissue hyperplasia invades the organ, becoming especially marked in the interior of the lobules and in the fissure. In time this fibrous tissue undergoes contraction and the liver becomes reduced in size and hard, very much as it does in hepatic cirrhosis. The once enlarged and chronically congested organ shrinks, becoming dense and thin bordered, more or less granular, but still fairly regular in outline, excepting that its notch is greatly exaggerated. According to Eisenmenger, it is by the contraction of the connective tissue within the fissure of the liver and consequent compression of the portal vessels that the shrinkage in the size of the organ leads to ascites, the same as does the atrophy in Laennec's hepatic cirrhosis.

Finally, the adherent pericardium may exist without chronic inflammatory changes in the endocardium or myocardium, but in most instances it is associated with valvular disease, or with chronic myocarditis.

SYMPTOMS.

These depend not only on the extent and situation of the pericardial adhesions, but also upon the co-existence, or not, of other lesions as valvular defects. If the two layers of the sac alone are adherent, and particu-

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