was distended in 2 of the 6 patients by the same method and the resultant signs and symptoms similarly recorded.

Our observations seem to indicate that: 1. Distension of the *gallbladder* gives rise to deep epigastric discomfort more severe but similar to the attacks of indigestion in gallbladder disease. 2. The discomfort is not referred to the gallbladder region. 3. Distension of the gallbladder does not cause nausea and vomiting. 4. Distension of the *common duct* is much more painful than distension of the gallbladder and in addition is accompanied by nausea and vomiting. 5. Objectively and subjectively the patients have respiratory difficulty when the gallbladder or common duct is distended, especially inspiratory difficulty. 6. It was found impossible to reproduce the usual referred pain in the back, infrascapular region or the right upper quadrant. 7. The pain was not altered in one case in which the right upper quadrant was novocainized. 8. Distension of the gallbladder, producing contact with the parietal peritoneum, gave localized pain. This was markedly relieved by infiltration of the overlying abdominal wall with novocaine.

Since referred pain could not be produced by mechanical distension, weight is given to the argument that referred somatic pain and tenderness are more likely to result from an inflammatory lesion caused by a "peritoneocutaneous" reflex instead of a "viscerocutaneous" reflex. It is indicated that surgically it might be well to explore the common duct in those cases in which vomiting has been a prominent symptom, regardless of the history of jaundice or size of the common duct.

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Pathogenesis of Trichinous Myocarditis.

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Several authors have recognized and described the histopathology of the parenchymatous lesions and accompanying myocarditis which are seen in the hearts of human cases of trichinosis dying in the fourth to eighth week of the disease. The importance of these lesions in the production of the circulatory failure, which is the cause of death in these cases, has been emphasized.[1] All who have

[1] Weller, C. V., and Shaw, M., *Trans. Assn. Am. Physicians*, 1932, **47**, 41.

had opportunity to study such material have noted the absence of encysted larvae in the heart muscle even though the skeletal muscles showed at the same time very many encysting and encysted organisms. Almost all of those who have searched sections prepared from such hearts have failed to find larvae present at all. Only Zenker[2] and Frothingham[3] saw them in the human heart, but Graham,[4] working with rats, described lesions similar to those in human cases and in addition found embryos associated with the inflammatory foci. This complete absence of encysted forms with but meager evidence even of the presence of larvae, has led to uncertainty as to the pathogenesis. Simmonds[5] concluded that the myocarditis was due to a toxin produced by the trichinae and brought to the heart in the circulating blood.

In the present study, series of white rats were fed, in approximately known numbers, the larvae digested (McCoy[6]) from trichinous meat. Animals were killed at appropriate intervals and both fresh, teased preparations and stained sections were made from their tissues. The myocardium showed alterative and exudative lesions in all respects comparable to those found in human hearts. Trichina embryos were found in these foci as early as 5 days after feeding and for some time thereafter. In rats examined after active migration of larvae had ceased, the myocardium showed no reaction, although encystment of larvae in skeletal muscle was occurring to a marked degree. If the myocarditis were of toxic origin it should not have subsided during this stage. Thus by 2 lines of evidence it appears that it is the presence of the larvae in the myocardium and their active migration and not a blood-borne toxic substance which produces the characteristic myocarditis. It is not clear what are the factors, probably inherent in the heart muscle, which prevent encystment.

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[2] Zenker, F. A., *Arch. f. path. Anat. u. Physiol.*, 1860, **18**, 561.

[3] Frothingham, C., *J. Med. Research*, 1906, **15**, 483.

[4] Graham, J. Y., *Arch. f. mikr. Anat.*, 1897, **50**, 219.

[5] Simmonds, M., *Centralbl. f. allg. Path. u. path. Anat.*, 1919, **30**, 1.

[6] McCoy, O. R., *Am. J. Hyg.*, 1931, **14**, 484.