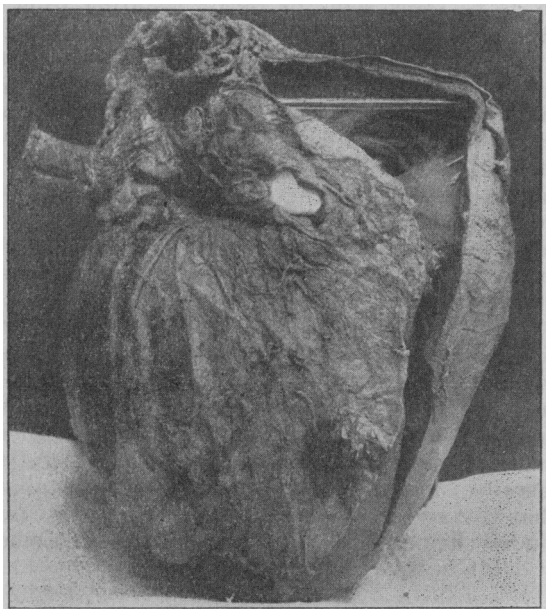


made previous to the autopsy showed that it appeared very much sooner than is usual.

Pleural cavities: The right lung was adherent to the chest wall at the upper and outer portion of the lower lobe. The left pleural cavity showed no adhesions, but was filled with liquid blood; the lung, which was half the size of the normal organ, being crowded upwards and to the right. Cross section of either lung revealed no pathological condition.

Heart and pericardium: The heart filled the entire pericardial sac and the pericardium was adherent over the greater surface of both auricles and ventricles, the pericardial cavity being practically obliterated. The heart was greatly hypertrophied and it, together with the aortic arch, pericardium, trachea and great blood vessels at the base, weighed 2 lb. 6½ oz. The walls of both ventricles were greatly hypertrophied, the wall of the left ventricle being 1½ in. in thickness, while that of the right was ¾ of an inch thick. The left auriculoventricular valves were calcareous and shrunken but presented no vegetations. A similar condition was found in the valves guarding the tricuspid opening. Both auriculoventricular rings were markedly stretched. The right ventricle and left auricle were enlarged and dilated. The right auricle was greatly hypertrophied and encroached upon the territory normally occupied by the left auricle, so that it occupied the greater part of the posterior surface of the base. The wall was thickened in some places, but at one point it was of paper thickness and presented an irregular tear, 1¼ in. in length, which extended through the myocardium, pericardium, and the attenuated reflection of the posterior mediastinum. Rupture had occurred into the left pleural cavity, a result that would have been impossible had not the right auricle occupied the greater part of the posterior surface of the base. There was no clot in the external opening of the wound. The semilunar valves guarding the aortic orifice were markedly thickened, shrunken, and adherent; they presented calcified vegetations. The ascending por-



Rupture in posterior wall of right auricle.

tion of the arch was slightly dilated. The pulmonary valves were normal. The *cor bovinum* had resulted from an aortic stenosis and regurgitation.

The coronary arteries were pervious to a probe

although both were atheromatous and the lumen of the left was markedly diminished in calibre at places.

Microscopical examination of specimens of the cardiac muscle was made by Dr. F. T. Fulton, pathologist of the Rhode Island Hospital, and the following is an epitome of his report:

Section of muscle from right auricle shows no evidence of fragmentation. The nuclei of the muscle fibers are distinctly swollen, being occasionally more than twice the normal size, rather pale and finely granular, the chromatin being uniformly distributed throughout the nucleus. Section from the left ventricle shows uniform enlargement of the nuclei. The muscle fibers are wavy in outline and the transverse striations are often very indistinct although in some places they are fairly well marked. Section from the left coronary artery shows the intima to be irregularly thickened, being in many places much thicker than the media of the artery. The tissues was not examined for fatty degeneration because it was not received until after it was hardened in formalin.

Remarks. — The above case is unique in several ways: Rupture into the pleural cavity rarely occurs and was probably determined by the obliteration of the pericardial cavity through the formation of adhesions. The patient was only thirty-six years old, and it is unusual for spontaneous rupture to occur in one of his years. The case presented the physical signs and symptoms of aortic aneurysm, and was accompanied by symptoms of pressure upon the left recurrent laryngeal nerve. The slight enlargement of the ascending portion of the aortic arch present could not have possibly caused this clinical picture. Lastly, the chronic arteritis was advanced in the coronary arteries while the systemic vessels were quite normal.

I wish to thank Dr. F. T. Fulton for valuable aid received in the preparation of this article, by his kindness in making and reporting upon the microscopical examination.

BIBLIOGRAPHY.

- Meyer: *Deutsches Archiv. für Klinische Medizin*, 1888, Vol. xliii, p. 379.
 Elleaume: *Essai sur les Ruptures du Cœur. Thèse de Paris*, 1857.
 Councilman: *Boston Medical and Surgical Journal*, 1893, Vol. cxxix, p. 457.
 Shelby: *Medical Record*, Aug. 28, 1897, Vol. lii, p. 319.
 Amyot: *Canadian Practitioner*, Vol. xxiv, p. 441.
 Greig: *Canadian Practitioner*, February, 1898, Vol. xxiii.
 Böttger: *Archiv. der Heilkunde*, 1863.
 Barth: *De la Rupture Spontanée du Cœur. Archives Génér. de Médecine*, March, 1871.
 Moore: *Transactions of the Pathological Society of London*, Vol. xlv, p. 24.
 Green: *British Medical Journal*, June, 1895, p. 1202.
 Noble: *Ibid.*, 1889, Vol. i, p. 77.
 Williams: *Lancet*, Dec. 12, 1896, p. 1687.
 McIntosh: *Ibid.*, 1896, Vol. i, p. 239.
 Mickle: *Edinburgh Medical Journal*, February, 1884.
 Hamilton: *Philadelphia Medical Journal*, Jan. 24, 1903, p. 173.
 Sheldon: *Medical Record*, Nov. 5, 1904, p. 737.
 Osler: *Practice of Medicine*, Fifth edition, p. 753.
 Bruce: *Journal of Mental Science*, 1892, Vol. xxxviii, p. 85.
 Quain: *Lumelian Lectures. Lancet*, 1872, Vol. i.

SPONTANEOUS RUPTURE OF THE HEART IN A CASE OF SENILE DEMENTIA.

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THE following case is reported owing to the infrequency with which such cases come to autopsy.

Clinical summary: T. E., male; eighty-three years of age; admitted to Taunton Insane Hospital, Jan. 11,

1906. No reliable previous history obtainable. Said to have been insane for one year; became talkative, abusive, quarrelsome and showed increasing motor and psychomotor activity. Spoke of spirits following him, threatening him injury. Recent memory defective.

A well-developed old man looking not more than seventy-five years of age. Together with the usual senile traits he had a slight deviation of the tongue to the right, but no other focalizing symptoms. No eye symptoms; reacting pupils; nothing of importance in the deep reflexes. Slight dribbling of urine, the urinary examination showing only a trace of albumen, a few pus corpuscles and acid reaction. Heart's dullness slightly increased to the left; heart's action irregular, no distinct murmurs. Peripheral arteries hardened. Radial pulse weak and thready.

During his hospital residence, he was much excited, constantly producing, with loud voice, an incoherent stream of talk, about murders, shooting, etc. Evident auditory hallucinations. He was very irritable; disoriented; recent and remote memory defective. Slept very little. When awake was in almost continuous motor activity. He never complained of cardiac distress. His condition varied little.

The night of Jan. 28 he was seen by the night nurse at twelve o'clock and was in no way different from usual. Twenty minutes later he was found dead in an easy position in bed, not having been seen in the meantime.

The autopsy was performed thirteen hours after death.

Anatomical Diagnosis: Hemorrhage into pericardial sac from rupture of the heart muscle near tip of left ventricle; patch of anemic necrosis at apex of left ventricle; sclerosis of coronary arteries with a thrombus in branch near necrotic area. No significant sclerosis in aorta, but sclerosis in basal cerebral vessels and peripheral arteries; chronic fibroid pleuritis; biliary calculi; calculus in urinary bladder, with slight subacute cystitis; pachymeningitis chronica; leptomeningitis chronica; small subdural clot, partially organized in right parietal region; hydrocephalus ex vacuo; atrophy of frontal lobes; *état criblé* in basal gray nuclei.

I shall describe in detail only the findings in connection with the circulatory system which have a direct bearing upon the subject in hand.

The following is from the autopsy protocol: "Upon opening the pericardium, which is tense and bulging, there is a gush of dark fluid blood, 500 cc. There are a few small dark clots. On examination an irregular tear is discovered on the anterior aspect, 8 mm. in length, parallel with the long axis of the heart. The lower edge is 15 mm. from the apex and it appeared superficially to be connected with the right ventricle being situated immediately over the right ventricular wall. Further examination shows that the external opening is connected by a channel with an area of degeneration at the apex of the left ventricle. This degenerated area is irregular in outline, somewhat wedge shaped, the greater part of the tissue darkish gray and of a necrotic parboiled appearance, but there are areas of healthy looking muscle intervening so that the necrotic patch is not sharply outlined from the normal muscle. It is softer than the surrounding muscle. The periphery of the patch has a congested appearance. The tissues about the channel are soft and infiltrated with blood. The channel from a position a little beyond the external part of the area of degeneration to the outer opening is covered only by the epicardium. Within this channel are a few dark clots and small grayish necrotic masses.

"The coronary arteries are decidedly thickened and show many raised yellowish patches without, however,

any calcareous formation. A thrombus is found in the coronary branch (anterior coronary) near the necrotic area. The myocardium is slightly pale but the acetic acid test shows no increase of fat. Heart weight, 370 gm. Valves normal, except aortic segments which are a little stiffened but without calcareous plates."

Microscopical examination of the coronary arteries show well advanced changes of arteriosclerosis with retrogressive changes in the thickened intima layer. Numerous hyaline spaces are seen and again necrotic patches extending into the media. The lumen is narrowed, irregular in outline. There is a characteristic subendothelial connective tissue increase. The endothelial lining has nowhere remained intact. The connective tissue cells are relatively scarce. There is an occasional round-celled infiltrate surrounding necrotic masses. Elastic layer split up.

A section from the center of the area of degeneration in the left ventricle shows a homogeneous poorly stained mass with very little indication of muscle fibers. There are quite numerous normal, and a few degenerated red blood corpuscles; scattered white cells, the majority of which are well stained; some pigment granules. Sections from the periphery of this area reveal muscle fibers in various stages of degeneration. Evidence of regeneration is shown by the presence of various forms of granulation tissue cells, situated between the degenerated muscle fibers.

The left ventricular wall at the upper part of the ventricle shows a slight increase of interstitial tissue, a distinct increase in brownish pigment around the poles of the nuclei, but very little thinning of the individual muscle fibers. The smaller vessels in this section show the indications of the arterial disease.

We have, therefore, in a man eighty-three years of age, a spontaneous rupture of the heart as a result of myomalacia cordis, the accident being determined by mental and physical excitement. The infarction which had existed sufficiently long so as to show signs of repair resulting from a thrombus in the anterior coronary artery.

The most noteworthy feature of the case is its rarity. Krouskoff reports only three cases in eight thousand autopsies. The site of the rupture in relation to the area of degeneration at the apex of the left ventricle is somewhat unusual, yet it can be correctly designated as a rupture of the left ventricle near the tip, a common location. Odriozola reports rupture of the left ventricle in 96 out of 132 cases which he collected from the literature.

The age of the patient, the condition of the arterial system, were favoring conditions almost invariably cited in cases of spontaneous heart rupture. The pre-existing myocardial disease, anemic necrosis, is regarded as a less frequent cause of heart rupture than other degenerative diseases of the myocardium. It is unfortunate that the accident occurred at a time which precluded the possibility of observation, as the mode of death in these cases is of interest.

TYPHUS FEVER IN PHILADELPHIA.—A case of typhus fever has been discovered in Philadelphia in the person of a young man recently arrived from Denmark. The patient was removed to the Municipal Hospital, where a fatal termination ensued. This is the first case of the disease in Philadelphia since 1888. — *Medical Record*.