ruary, 1907. This abscess had to be lanced several times and required four months for its cure, during which time she had chills and fever. In the following August she suffered from pain in the bladder and frequent micturition, noticing several times slight discoloration of the urine with blood. December 12 pain in the bladder was severe and she expelled from it several ounces of pus with entire relief. In January, 1908, and each month afterward, she suffered a similar attack. Frequently after August, 1907, she had noticed blood but not pus in the urine. Early in October, 1908, she suffered from another attack of chills and fever and noticed a swelling in the left side of the abdomen, and that while lying on the bedpan and pressing on the swelling pus would escape from the bladder with the urine. The patient thought that she had lost fifty pounds in weight since the birth of her child.

Examination.—This revealed an enlargement about six inches in length in its longest diameter in the region of the kidney and, extending from it to the bladder, the ureter was enlarged to about one and a half inches in diameter throughout its entire course. The bladder wall was much thickened. Urinalysis showed a large amount of pus, which on segregation, was limited to the affected side. Several bacteriologic examinations failed to demonstrate the presence of tubercle bacilli. Nevertheless, the diagnosis of tuberculosis of the left kidney, ureter and bladder was made.

Operation.—On Nov. 6, 1908, the kidney and ureter of the left side were removed by the method already mentioned. Longitudinal incision of the kidney after its removal showed at least ten pockets filled with thick blue pus; the ureter contained pus of the same appearance. Two sections of the kidney were examined by the pathologist who reported negatively as to tuberculosis both as to tissue and contained pus. He considered it a picture of pyelonephritic abscess formation and purulent infiltration.

The patient made a very good recovery.

CASE 3.—*Patient.*—Mrs. A., aged 25, Russian, secundipara, admitted to Columbia Hospital for Women, Dec. 3, 1907, for pain in the right side and irritability of the bladder. She had passed bloody urine and had missed three menstrual periods.

Examination.—A cystoscopic examination found the bladder mucosa markedly reddened, the bladder wall thickened, both ureters palpable through the vagina, the right being the size of a lead pencil and hard and the left smaller and not so distinct. Through the abdominal wall the right could be felt all the way to the kidney, the left was not traceable in this manner. A mass was felt in the region of the right kidney of about twice the size of that organ, as the uterus was considerably enlarged pregnancy was diagnosed, she was kept under observation until Jan. 11, 1908, when she left the hospital to which she returned at the time of the birth of her child, May 15.

She was readmitted to the hospital Oct. 20, 1908, suffering with pain in her right side, in the bladder and frequent and painful micturition. The abdominal mass had now increased to a size in excess of that of the head of a new-born infant.

Operation.—Examinations in this instance were negative as to tuberculosis, and on November 10, 1908, complete nephroureterectomy was done by the method described in this paper. The right kidney, about 10 inches in length and about 12 inches in transverse circumference, contained many separate abscesses beside purulent inflammation of the calyces and pelvis of the kidney. The pus was very thick, and in some instances granules of a pale blue color were noted. No calculi were found. The ureter was 1/2 to 3/4 inches in diameter, though having but little more than usual measurements at the lower end and one inch in diameter at the upper end. The transverse incision was directly opposite the umbilicus and about six inches in length.

Recovery.

CASE 4.—*Patient.*—M. B., aged 29, colored, multipara, was admitted to Columbia Hospital for Women, Jan. 4, 1909. She was suffering from frequent micturition attended by pain at the beginning and ending of the act, which had existed nine months. Blood had usually been noticed in the urine during the past month.

Examination.—The left kidney and ureter were slightly enlarged, the bladder thickened and inflamed and tubercle bacilli were found in the urine from that side. The daily quantity was 705 c.c.

Operation.—Jan. 19, 1909, nephroureterectomy was performed by loin extraperitoneal and vaginal routes. The peritoneum was opened for a half inch accidentally and inspected near the opening. It appeared normal and the wound was at once closed. A thickened and nodular ureter and a pyonephrotic kidney were removed in one mass and without leakage.

The patient gradually failed and died March 1, 1909. At an incomplete necropsy next day the following points were noted.

Postmortem Examination.—The parietal and visceral peritoneum were generally and thickly studded with small, white, pinhead-sized tubercles which were most numerous around the area from which the kidney had been removed and the adjacent coils of intestine. Adhesions were rather numerous and firm. A small quantity of yellow pus was present in the pouch of Douglas. The right kidney, small, soft, and somewhat injected, appeared to have undergone some parenchymatous degeneration, but there were no gross lesions of tuberculosis. The walls of the ureter were not thickened. The bladder wall was thickened and the interior studded with older tubercles and ulcers. The lungs, liver, heart and spleen were all pale, soft and bloodless, in harmony with the emaciation presented.

The Rochambeau.

CARDIAC THROMBOSIS

THE CLINICAL AND PATHOLOGIC FINDINGS IN THREE CASES *

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There has been but little added to the literature of true heart thrombi since the very complete monographs of Robinson and Welch. Desultory reports of such conditions are not uncommon, but, as Welch has pointed out, many of the observations on so-called heart thrombi are really records of postmortem heart clots entangled in, but not organically attached to, the muscle columns and trabeculae. True heart thrombi, whether of the pedunculated or ball variety, are of such uncommon occurrence that their occasional incidence would seem to warrant study of the clinical and pathologic sides of cases in which they have been found postmortem. And this seems especially important in so far as the possibility of antemortem diagnosis carries with it a not altogether worthless bearing on the prognosis in a given case. It does not appear necessary here to point out the practical value of antemortem diagnosis of this condition with regard to the application of therapeutic measures.

The term "cardiac thrombus" must be reserved for a solid or partly solid structure, primarily formed from blood elements, which develops in one or more chambers of the heart during life. Such a mass may be attached to the cardiac wall by a more or less altered base, or may exist as a free foreign body within a heart cavity. When cardiac thrombi are measured by this standard they are uncommon postmortem findings. This applies especially to those of the free, or "ball" variety.

Of the pedunculated variety of cardiac thrombus, Welch was able to discover thirty-three cases in 1899, while a much smaller number of "ball thrombi" are on record. Flat, broad-based, mycotic thrombi are more common. They may be the beginning of either of the two varieties mentioned.

The mode of formation of heart thrombi has been long a subject of difference. The early false conception

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of what was really meant by true thrombi was doubtless responsible for much confusion. The closer study of recent cases, from histologic standpoints, coupled with added knowledge of the chemical interaction of certain constituents of the blood, has elucidated some of the problems which vexed the early investigators. It is now commonly accepted that while in the production of a thrombus the individual exciting causes may vary widely, there are certain basic principles to which thrombus formation within the heart conforms. The first of these principles is that some damage done to the endocardium is concerned. This may be the result of bacterial, toxic or purely mechanical agents. Whatever the individual cause, the liberation of so-called thrombokinase, acting on the thrombogen in the circulating blood, with the formation of more or less fibrin, results in blood-clotting. This clot may be minutely local or early involve gross areas. The second factor in production of thrombi appears to be that of an increase of the hemagglutinins in the blood stream. This may be brought about by bacteria, either locally involving the endocardium or freely circulating in the blood stream. It would also seem that the introduction of certain proteid poisons into the circulation might be responsible for increased agglutination of red cells, with thrombus formation. In the light of recent researches, it does not seem that the increase of blood platelets in itself has any definite bearing on either agglutination or blood coagulation. It is not impossible, however, that development of necrotic foci locally in the endocardium as a result of microbic invasion might release into the blood stream hemolytic toxins, the result of which would be an increase of the thrombokinase in the heart cavities with coincident blood clotting by acting on blood elements. The third factor in the production of thrombi is that of slowing of the blood stream. Changes in direction of the stream with the formation of eddies may also have some bearing on the formation of thrombi in certain definite localities, as, for example, in the auricular appendages or between the muscle columns of the ventricles.

It might be of value to consider briefly the bearing of certain intracardiac and systemic conditions on the production and development of heart thrombi. The condition appears to be relatively infrequent in the majority of infectious diseases. It would seem, from tabulated statistics from various autopsy records in this and other countries, that, with the exception of influenza, tuberculosis, and perhaps rheumatism, cardiac thrombi are extremely uncommon findings in infectious disease. There is reason to doubt, however, the statement that cardiac thrombi are a rarity in syphilis, as I shall attempt to show later. Certain blood states are occasionally complicated by cardiac thrombosis, as, for example, chlorosis, leukemia and pernicious anemia. In the cachexias resulting from cancer (chronic), malaria, and the like, thrombi sometimes occur. Valvular lesions of the heart itself are usually secondary to infective processes in the endocardium. There is not uncommonly development of thrombi from such with vegetations as primary origin. Their organization and extension or contraction may be responsible for the valvular inefficiency or for insufficiency of the myocardium itself. Some writers have attempted from the meager statistics at hand to show the definite relationship of the development of heart thrombi to certain valvular disturbances. The much-quoted tables of Pawlowski would seem to demonstrate thrombosis of the left auricle was about five times as common as that of the right, and about ten times as common as in the left ventricle. Primary thrombosis of the right ventricle

appears to be unknown. The usual cardiac finding, apart from general muscular insufficiency, particularly localized in one chamber wall, appears to be mitral stenosis. This applies particularly to cases of "ball thrombi." It is to be readily appreciated how obstruction at the mitral orifice mechanically alters the left auricular circulation, that chamber being especially suited to early sluggishness of its blood content. It is not infrequent, however, to find heart thrombi in the left auricle where there is no disabement of the mitral orifice—indeed, no apparent primary cardiac lesion whatever.

The thrombi are variously located. The majority spring from the septum, the foramen ovale, and the auricular appendage. It is possible, however, to have their bases at any part of the endocardium. The valve flaps do not escape. Thrombi formed with infectious vegetations as their bases may thus seriously interfere with the function of the valve flaps and aggravate, mechanically, a lesion otherwise of but moderate degree.

The condition of the arterial tree in general doubtless influences the formation of thrombi in the heart. This is especially true where a high grade of sclerosis exists generally, with the presence of atheromatous patches on the endocardium. It is to be expected, then, that the majority of cardiac thrombi are found in patients past the middle of life. This rule is not, however, absolute. Pawlowski's table shows two cases of pedunculated thrombi in patients below the age of 20, and Keating mentions the occurrence of thrombi in children afflicted with various infectious diseases.

The symptomatology of cardiac thrombosis varies with the location of the foreign growth within the heart. It is commonly admitted that, while the condition may be suspected during life, it can not be accurately diagnosed. Several authorities, notably Laennec, have held, however, that when in the course of a cardiac lesion the signs become anomalous and confused, with practical loss of the clinical picture of either definite cardiac lesion or typical cardiac inadequacy, one should suspect the possibility of thrombus formation. The signs and symptoms are those of impeded circulation. These seem to be most marked when the thrombus is located in the auricles. The thrombi located here are apt to be larger and to extend through the cardiac orifices into neighboring chambers. The inability of the more or less weak auricle wall to resist back-flow of blood and dilatation soon brings about pronounced alterations in the general circulation.

The three cases furnishing the basis of this communication occurred during the past two years at the University Hospital (Professor Dock's service). The cases were all of such interest that one might be pardoned submitting a rather full report of them. Of the three cases, the lesion was correctly diagnosed antemortem in one. Two patients were males, one female. Two were negroes. The ages were, respectively, 43, 56 and 60 years. The three cases exhibited, clinically, cardiac valvular lesions, the mitral flaps appearing to be the seat of the primary process. The reasons given for entering the hospital were shortness of breath, pain about the heart and the region of the stomach, general weakness, cough, with sputum, cyanosis, edema, and palpitation.

REPORTS OF CASES

CASE 1.—Patient.—L. J., negro, aged 43, gardener, entered the hospital, Out-Patient Medical Clinic, May 23, 1907, on account of extreme shortness of breath, precordial pain and distress and weakness.

History.—As is frequently the case in these patients, little could be obtained. The patient had had gonorrhea several times and there was a strong suspicion of lues. He had been

well up to within two months of coming to the clinic. He then began to note weakness on exertion, shortness of breath and cough. There was abdominal discomfort in the left lower quadrant of the abdomen, although his bowels were regular. His appetite became poor and there was a tendency to belching of gas.

Examinations.—The dispensary examination revealed a rather well-built man with cyanosis of moderate grade at the ears, lips and finger tips. The heart was slightly enlarged in all directions. At the apex, the first sound was impure, and the second roughened. Both tricuspid sounds were impure, but not so harsh as the mitral sounds; the second pulmonic was very sharp and rough; there was a soft systolic bruit over the aortic area; the heart rhythm was very irregular; there was moderate sclerosis of the radials; the pulse was somewhat small, irregular and weak. The blood pressure, registered by the Stanton (12 cm. cuff) systolic 165 mm. Hg., diastolic, 118 mm. Hg. The lungs were negative.

The patient was placed on a mild diuretic and given saline cathartics. He left and was lost sight of for about a month, when he again came for examination. The shortness of breath, especially on exertion, was pronounced; there was great weakness and cough. The heart examination revealed no greater enlargement than when the patient was first seen. The sounds at the apex were now clear and distinct, and no murmurs were heard, although there was roughening of the mitral second, and

pallor; large drops of sweat stood on his forehead; the extremities were moist, without edema; the pupils were equally dilated, but sluggish.

Heart: Apex in fifth space, just outside the mid-clavicular line. The right border extended slightly beyond the right edge of the sternum at the fourth right interspace; the right border could not be made out higher up. The upper boundary on the left was at the upper edge of the fourth rib, in the left parasternal line; the left edge was made out about 1 cm. outside the mid-clavicular line in the fifth left space. (Fig. 1.) On auscultation the sounds were very weak at the apex; the first was murmurish. The second pulmonic was accentuated and divided. It was very difficult to make out the sounds over the base; the aortic sounds were very weak. There was moderate arrhythmia, with occasional extrasystole. The radial pulse was very small, irregular, but coincident with the heart beat. The vessels of the neck, especially on the right, were moderately engorged, but positive pulsations were not noted in the jugulars. The blood pressure was 116 (Stanton, 12 cm. cuff). Liver and spleen not enlarged.

Lungs: Pleuritic friction in the left axilla and back, with diminished resonance in both lower backs, more on the left.

Abdomen: Tense and difficult to examine, especially full in the mid-epigastrium; deep palpation impossible on account of muscle spasm throughout; there was pain on moderate pressure in the epigastrium and in the left lower quadrant. Catheterization revealed stricture in deep urethra.

Fig. 1.—Case 1: Outlines of percussion dulness when patient entered hospital. L.B.H., left border of heart; A., apex beat; S.D., splenic dulness; L.D., liver dulness; R.B.H., right border of heart.

both sounds were astonishingly sharp. There was noticeable accentuation of the second pulmonic sound, and a faintly heard systolic bruit was made out at the aortic area. The heart exhibited marked arrhythmia; there was no peripheral edema, nor enlargement of the liver or spleen. The pulse was small and very irregular. Strychnin and Carlsbad salts were ordered and rest insisted on. The patient then disappeared for about ten days.

At his third appearance it was evident that he was in great distress. The cyanosis, cough, dyspnea, abdominal pain and weakness were extreme. He was ordered into the medical ward. At entry there were no murmurs heard over the precordia. The aortic sounds were very weak; the heart was somewhat enlarged in both directions (Dr. Van Zwaluwenburg); the systolic blood pressure registered with the Stanton (12 cm. cuff) 115 mm. Hg.

The patient was put to bed and his condition remained unchanged for twenty-four hours. At that time I was called to see him on account of his complaining of great pain in breathing, across the mid-epigastrium and the lower left axilla. He lay in the passive dorsal position, with eyes partly closed; his expression was anxious in the extreme; lips and ears were slightly cyanosed, but presented rather a striking degree of

Fig. 2.—Case 1: Outlines of percussion dulness two days before death. (Postmortem: Thrombosis of right and left auricles.) L.B.H., left heart border; A., apex; S.D., splenic dulness; L.D., liver dulness; R.C.D., right cardiac outline to right of sternum.

The man was given morphin, enemata, and cathartics; ice was placed locally over precordia and left thorax. The following day his temperature rose to 101 F. in the morning and restlessness was extreme. It was impossible to keep the man in bed, the greatest relief being when he was walking about. His expression was frightened; his face and neck a dusky pallor and covered with huge drops of cold sweat; breathing audible and of the Cheyne-Stokes type. Bowels had not moved in spite of cathartics and enemata. Dyspnea and abdominal pain were marked. The breathing was distinctly audible throughout the ward.

The heart showed distinct changes from the day previous. (Fig. 1.) The apex beat could neither be seen nor felt. By auscultation it was made out in the fifth left space, just within the left mid-clavicular line. The heart had, however, greatly enlarged towards the right, and the enlargement was of peculiar outline, when associated with other clinical findings. In the fifth right space the right cardiac border could be made out nearly three fingers' breadths to the right of the sternum. The line of dulness extended upwards at about the same distance from the right edge of the sternum almost to the second rib, where it curved rather sharply inward to meet

the sternum at the angle of Ludwig. There was slight outward bulging in the second and third spaces. This atypical rightsided enlargement of cardiac dulness was not associated with noticeable venous engorgement of neck vessels and there was no positive venous pulsation in the jugulars. Liver and spleen were negative; there was no peripheral edema. (Fig. 2.) On auscultation, the sounds at the apex were weak. A soft, blowing, systolic bruit could, however, be made out at the mitral area, the systolic bruit being well heard in the axilla and the left back. Over the tricuspid area the murmur with the first sound became louder and more prolonged. It was carried well up the left edge of the sternum. At the third left costosternal junction there was a suggestion of soft, blowing, diastolic bruit. The sounds over the base were audible with difficulty. The second pulmonic was divided, and a late diastolic bruit was made out. In the aortic area the sounds were only occasionally audible. There was marked arrhythmia—a veritable "delirium cordis." The radial pulse was barely palpable, rather quick and did not appear to differ in rate with that of the heart. The lung examination revealed pleurisy on the left

firmer than in others. The base of this clot was adherent to the wall of the right auricle, filling the ear. It was dark yellow, tough and fibrous. On cutting into this base there were cyst spaces filled with broken-down and altered blood. These older clots were gradually merged into more recent clots extending continuously through the tricuspid ring. (Fig. 3.) Left heart: In the left auricle was a large currant-jelly clot attached to a firmer organized clot in two places. The main attachment was in the ear of the auricle, and the smaller over the posterior wall. There was a rather recent thrombus at the mitral ring. Just between the mitral flaps was a firm white clot, entangled between the chordæ tendineæ and attached to the clot in the auricle. This was continuous to a clot extending to the aortic ring. At the base of the clot on the mitral valve was an area of sclerotic endocardium. The chordæ tendineæ were not retracted or thickened. The ventricular wall was light brownish-red, slightly cloudy and firm; there was slight fatty degeneration. (Fig. 3.)

Lungs: Recent hemorrhagic infarcts; (embolic) passive congestion and edema.

Pulmonary vessels: Organizing thrombi.

Liver: Extreme passive congestion; nutmeg liver and icterus.

Kidneys: Cloudy swelling; small healed anemic infarcts; recent sclerosis of renal vessels.

Adrenals: Chronic passive congestion; excessive fatty degeneration.

Fig. 3.—Case 1: Heart showing thrombus in right auricle extending through the tricuspid orifice into the right ventricle. (From the Pathological Museum of the University of Michigan, Specimen No. H. 46. Courtesy of Dr. Aldred Scott Warthin, Director.)

with airless tissue in the back. The abdomen was still rigid and tender. The bowels could not be made to respond to purgatives or enemata.

The condition described continued about the same for another day and then the patient suddenly expired while being bathed.

Postmortem Examination (Prof. A. S. Warthin, prosecutor).—Seven hours after death.

Clinical Diagnosis: Mitral, tricuspid and aortic regurgitation; suspected thrombosis of right auricle; pleurisy, pneumonia (terminal); nephritis; cystitis; intestinal obstruction; infarction.

Pathologic Findings: Heart: Subepicardial fat increased; small subepicardial hemorrhage on anterior surface; few on posterior surface; ventricles contracted; both auricles filled with currant-jelly clot. Right heart: Auricle enormously dilated; extended about four fingers' breadths beyond right sternal margin; muscle fibers separated. Ventricle wall measured 4 to 6 mm. On opening the right ventricle, there was seen a clot extending through the tricuspid orifice, in parts

Fig. 4.—Case 2: Outlines of percussion dulness. (Postmortem: Thrombosis of left ventricle.) L.H.B., left heart border; A, apex beat; S.D., splenic dulness; L.D., liver dulness; R.H.B., right heart border.

Pancreas: Atrophy; sclerosis of vessels; some fatty infiltration.

Prostate: Chronic prostatitis.

Spleen: Chronic passive congestion; anemic infarction.

Pathologic Diagnosis.—Summary: Cardiac thrombosis; chronic interstitial myocarditis; obliterative endocarditis, probably syphilitic; arteriosclerosis; thrombosis of pulmonary vessels; hemorrhagic infarcts of lungs; anemic infarcts of spleen and kidneys; chronic passive congestion of all organs.

CASE 2.—Patient.—J. B., male, aged 56, merchant, American, was brought to the hospital by his brother on account of "heart disease," "Bright's disease" and addiction to morphin and possibly other drugs.

History.—Family history negative so far as could be determined. Patient had scarlet fever as a child; diphtheria at 15 (ill six weeks; delirious); malaria in southern Missouri, at 26, and attacks continued for some time after coming to Michigan; paroxysms every other day at first, then daily; "remittent fever" at 44; denied gonorrhea and lues. Took Keeley cure at Dwight two years previous to entering the hospital and had not taken intoxicants since. Previous to that time had been a hard drinker and had taken Keeley cure twice with relapses. He had a cough for fifteen years and at times it was very severe, the patient having raised much sputum, and

he said some of it was deeply blood-stained; of late had had "inflammation of the bladder" and incontinence of urine.

Examination.—A large, heavy man of asthmatic type. Skin of face sallow and dusky; slight cyanosis of lips, chin, ears, finger tips; slight edema of feet and legs; coughed frequently and raised mucopurulent sputum.

Thorax: Large, deep, short, broad. **Heart:** Diffuse pulsation in the region of the left nipple; apex beat not distinctly seen or felt. Dulness, on the fourth rib, outward to the mid-clavicular line, and inward to just beyond the right edge of the sternum, a total width of 14.5 cm. (Fig. 4.) **Auscultation:** Sounds very weak at apex; pulmonary second very weak; aortic second weak, with soft diastolic murmur (auscultation difficult on account of the harsh breathing; radials very small; and pulsations irregular. **Lungs:** Harsh breathing throughout, with occasional crackling rle; diminished resonance in the right back.

Liver dullness small.

Abdomen: Somewhat distended; dull in the dependent parts.

Blood pressure: Systolic, 152 mm. Hg.; diastolic, 108 mm. Hg. (Erlanger, 15 cm. cuff).

Urine: Small amounts of albumin with few granular and hyaline casts; pus with numerous cocci; few red blood cells. **Blood:** Reds, 5,280,000; whites, 8,600; Hg. 95 per cent. Tallquist.

Sputum: Thick, tenacious, mucopurulent, copious; many large cocci in pairs; many small cocci in groups; many polynuclear leucocytes; many "heart failure cells" and some red blood cells.

The patient remained under observation for about three months. At times there were periods of considerable comfort, with frequent relapses. He finally developed extreme myocardial inefficiency, which, coupled with infarction and edema of the lung, caused his death.

Postmortem Examination (Prof. A. S. Warthin, prosecutor).—Twelve hours after death.

Clinical Diagnosis: Dilatation of heart; stenosis and insufficiency of mitral valve; infarct of lung; hypostatic congestion; chronic bronchitis; chronic nephritis; nutmeg liver; terminal pneumonia?

Heart: Enlarged in all directions; dilated and hypertrophied; marked dilatation and hypertrophy of left ventricle; fibrous myocarditis and hemorrhagic infarction; sclerosis of coronaries, anemic infarction of ventricular wall, near the apex of the left ventricle. In the left ventricle was a moderately large thrombus, with organizing base, attached to the ventricle wall. The ventricle was greatly dilated, and there was relative insufficiency of the mitral valves. The endocardium showed atheromatous patches; the aorta was dilated and atheromatous; the aortic flaps were thickened and stiffened. (Fig. 5.)

Lungs: Chronic passive congestion; edema; old and fresh hemorrhagic infarcts; multiple emboli.

Spleen: Chronic passive congestion; atrophy; sclerosis.

Kidneys: Anemic infarct in stage of organization; chronic passive congestion; chronic parenchymatous nephritis; multiple embolic infarcts.

Adrenal: Congestion, with marked fatty change.

Liver: Nutmeg liver; early stage of cirrhosis (in part a central cirrhosis and in part of the atrophic type), small, miliary tubercles.

Testis: Edema; chronic congestion; small cysts in epididymis; calcified thrombus.

Pancreas: Fatty atrophy; increase of interstitial connective tissue; unusually large hyperemic islands of Langerhans.

Stomach: Chronic catarrhal gastritis.

Vessels: Sclerosis; atheroma.

Pathologic Diagnosis.—Summary: Cardiac thrombosis and infarction; cardiac hypertrophy; relative mitral insufficiency; chronic congestion of all organs; hemorrhagic infarction of lungs; anemic infarction of kidney; early stage of hepatic cirrhosis; atrophy of pancreas; arteriosclerosis and atheroma (syphilis?).

CASE 3.—**Patient.**—M. C., female, aged 60; negress, housewife, was sent to the hospital by her physician on account of shortness of breath, cardiac distress; chronic cough; swelling of feet and ankles and insomnia.

History.—Patient denies lues and gonorrhea; had not been ill much in life; always worked hard. About ten years ago she said she fell and broke leg; said she had had shortness of breath on exertion ever since; had had "weakness" in region of heart which prevented her moving about much or doing heavy work; about six months ago had a severe fright from house getting on fire. She became very short of breath afterward; felt very weak and could not lie down at night; ankles and legs began to swell; and recently thought that abdomen had become larger.

Examination.—Patient, a rather emaciated negress, propped up in bed, expression anxious; frequent cough; mucous rattle in throat; marked dyspnea; eyes injected; moderate cyanosis of lips, ears and extremities; tense edema to knees, with scaly eczema over legs; neck vessels injected and tortuous.

Thorax: Short, deep and broad; interspaces retracted during inspiration; thorax moved *en cuirasse*; right side below nipple much fuller than left; no Litten's shadow seen on either side; lung-liver border at seventh rib, mid-clavicular line. **Percussion:** Lungs, hyperresonance over both uppers with fair resonance in both axill. **Auscultation:** Large and small, moist and dry rles throughout, more especially on the left side. No alterations in voice transmission.

Fig. 5.—Case 2: Heart showing thrombus in left ventricle. (From the Pathological Museum of the University of Michigan. Specimen No. H. 65. Courtesy of Dr. Aldred Scott Warthin, Director.)

Heart: Apex beat in the fifth left space, 8.5 cm. from the median line; dullness extended two fingers' breadths to right of sternum in the fifth right space; one finger's breadth to right in fourth right space. The upper boundary of the cardiac dullness was at the upper edge of the fourth rib in the left parasternal line; the left edge was 11.5 cm. from the mid-line in the fourth left space. (Fig. 6.) There was noticeable pulsation in the middle line at the base of the xyphoid. **Auscultation:** First sound at apex was impure and occasionally divided; both features were more noticeable as one went toward the base of the xyphoid. The sounds over the base were very weak with the exception of the pulmonic second sound, which was moderately accentuated and murmurish. The radial pulse was small, quick, slightly irregular and compressible, with difficulty. The blood pressure was systolic, 242 mm. Hg.; diastolic, 168 mm. Hg. (Erlanger apparatus, 15 cm. cuff).

Abdomen: Rose three fingers' breadths above the level of the ribs; was rounded and circumference measured 92 cm. just below the navel. No pain points; no muscle spasm; no dullness in flanks. Liver, spleen and kidneys not felt. Glands negative throughout.

Urine: Slight traces of albumin, with small amounts of pus and blood.

Blood: Reds, 4,824,000; whites, 12,150; Hg. 95 per cent. (Tallq.).

Sputum: Yellow, ropy, thick and tenacious. Many polynuclear leucocytes and few heart-failure cells.

The patient was kept under observation about one month. She showed some improvement at the first part of her stay. Under appropriate medication, blood pressure became lower; orthopnea became less marked; the cardiac dullness diminished somewhat in size and the cough became less. About a week before her death she showed signs of failing cardiac action. Cheyne-Stokes breathing developed, edema of the lungs supervened and the patient died quietly. Towards the end of her illness she developed facial erysipelas. The heart again dilated, and a loud systolic bruit was audible at the apex. The sounds over the base continued weak. The neck veins became greatly engorged, particularly the left, external jugular, which pulsated noticeably.

Postmortem Examination (Prof. Aldred S. Warthin, prosecutor).—Performed eleven hours after death.

Clinical Diagnosis: Double mitral; cardiac dilatation and hypertrophy; tricuspid regurgitation; emphysema; pleural effusion (?), chronic congestion of kidneys; chronic interstitial nephritis; erysipelas facialis.

Heart: Pericardium adherent to border of left lung; tension greatly increased; 50 c.c. of turbid fluid in pericardial sac.

Fig. 6.—Case 3: Outlines of percussion dullness. Postmortem: "Buttonhole" mitral with thrombosis of left auricle.) L H B. left heart border; A, apex beat; S D, splenic dullness; L D, liver dullness; R B H, right heart dullness.

The heart was enormously enlarged; there was great arching of the septum to the left; the heart lay nearly transversely. The right auricle was greatly distended and formed about one-third of the entire heart size. The position of the heart was such as to rotate the pulmonary artery to an angle of 45 degrees. Between the pulmonary artery and the aorta was a thick, tendinous spot. The heart muscle was hypertrophic, with numerous atrophic fibers, however. It showed brown atrophy. The mitral valve showed an old sclerotic endocarditis. There was stenosis of the mitral flaps, of the true "buttonhole" type. In the left auricle was a moderately large organizing thrombus, attached by a broad base, at which point the endocardium was greatly thickened. (Fig. 7.) The right heart was markedly dilated, particularly the right auricle; the tricuspids were relatively insufficient. The aorta showed atheroma, marked sclerosis and calcification.

Liver: Extreme nutmeg liver; secondary cirrhosis.

Spleen: Atrophy and chronic passive congestion.

Lungs: Brown induration; edema; recent hemorrhagic infarcts; old calcified tubercle.

Kidney: Acute and chronic passive congestion; some atrophy and cloudy swelling; bacterial emboli in many of the small arterioles.

Adrenals: Passive congestion; atrophy.

Stomach and Intestine: Chronic passive congestion; stasis; gastritis and enteritis.

Uterine Wall: Fibroma.

Pathologic Diagnosis.—Summary: Erysipelas facialis; mitral stenosis; relative tricuspid regurgitation; thrombosis of auricle; dilatation of right heart; brown induration of lung; chronic passive congestion and atrophy of all organs; sclerosis and atheroma of aorta; healed tubercle in lung; slight goiter.

SUMMARY

Diagnosis.—The three cases here detailed present many features in common, while in certain respects they differ. The patients were past middle life. In all there was a fair possibility of syphilis. In each case the patient exhibited phenomena of cardiac insufficiency, in the main valvular. In all the cases, the mitral valves were affected. In but one was there stenosis. In this case, the stenosis was poorly manifested, clinically, with the exception of the early enlargement of the right side of the

Fig. 7.—Case 3: Heart showing "buttonhole" mitral orifice with thrombus in left auricle. (From the Pathological Museum of the University of Michigan. Specimen No. H. 71. Courtesy of Dr. Aldred Scott Warthin, Director.)

heart. In all the cases, postmortem examination showed the presence of emboli in various organs.

With respect to the thrombi themselves, a wide distribution is noted. In Case 1, the right heart, especially the auricle, was the primary seat of the thrombus. As circulation became interfered with, the mass extended into the right ventricle. There was, however, thrombus formation also in the left auricle, with a chicken-fat clot entangled in the chordae tendineae and interfering, at the end, with the free working of the aortic flaps. In Case 2, the ventricular wall gave rise to the thrombus. In this case, the signs of cardiac disease pointed strongly to the aortic valve being at fault, but postmortem the valve showed little change. In Case 3 is seen the rather typical, text-book heart thrombus, with mitral stenosis, impeded circulation in the left auricle, with pulmonary congestion and dyspnea early developing, together with

early enlargement of the right heart. The termination occurred with the dilatation of the right auricle after the tricuspid "safety valve" had thrown too great a strain on the weakened auricle.

Before closing I wish to call attention to certain aspects of Case 1 which rendered antemortem diagnosis of auricular thrombosis fairly possible. It will have been noted that the case first appeared with a somewhat atypical cardiac condition, namely, slight enlargement of the heart, but associated with extreme dyspnea, precordial distress and cyanosis, together with more or less pallor. Various murmurs and alterations in the character of the valve sounds were discovered, but neither murmurs themselves nor their location or propagation were definite enough to warrant the diagnosis of any particular valve lesion. After the patient entered the hospital, it was observed that prostration, precordial distress, restlessness and dyspnea, developed out of all proportion to the clinical findings in the heart itself. At the same time sudden, severe pains in the pleura, lungs and abdomen were noted—these strongly suggestive of infarction from emboli. As the case approached its termination, all the signs of failing cardiac action appeared from the point of view of the heart examination itself, but outside the heart the clinical condition was most atypical. The heart examination pointed strongly to mitral and tricuspid leakage, but extracardiac evidences were strangely lacking. Cyanosis was rarely marked; peripheral edema was extremely slight—indeed, absent till just before death; congestion of the liver, with liver pulse, was clinically absent. The spleen was not engorged. I would also call attention to the condition of the neck veins. At no time did they exhibit more than moderate engorgement, even when the signs of tricuspid insufficiency had been established for at least two days. There was absolutely no positive venous pulsation in either jugular vein, nor were positive venous pulsations noted in other veins, nor in the liver. I offer as explanation of this phenomenon the suggestion that the regurgitation wave from the right ventricle was changed in direction, or, indeed, broken up entirely, by its impinging on the large and constantly increasing heart-clot in the right auricle. So long as the auricle itself was able to propel a moderate amount of blood into the right ventricle, the circulation was maintained. It was only when the thrombus intruded on the right ventricle through the tricuspid orifice that stagnation became so great in the auricle as to bring on sudden cardiac dilatation and death. It does not appear to me that dilatation of the auricle along with sluggish blood stream could absorb, as it were, systolic pulsation from the right ventricle after the tricuspid had given way. This condition would be manifested by far greater venous congestion—in the neck veins and peripherally in the liver and extremities—than Case 1 showed. This is admirably demonstrated in Case 3, in which there was a buttonhole mitral with thrombus in the left auricle. Here the early dilatation of the right heart was promptly shown by peripheral edema, enlarged liver, and congested, pulsating veins in the neck, in addition to early enlargement of the right heart. It would seem to me, therefore, that the antemortem diagnosis of certain heart thrombi is not altogether impossible if the physical signs are carefully observed and recorded. And of these physical signs I would lay especial stress on atypical peripheral vascular manifestations with the more or less characteristic auscultation and percussion signs in the heart itself. The manifestations of embolic process should always furnish significant information.

With respect to the treatment of cardiac thrombi when the diagnosis appears likely, there can be little said. The question of how soon the case will terminate fatally is the leading one. The suspicion of a thrombus being present in the heart completely changes the picture from the prognostic standpoint, and certainly limits one's hopefulness with regard to whipping into line by drugs a heart whose compensation only appears to be failing.

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A NEW AND SATISFACTORY APPARATUS FOR ETHERIZATION IN OPERATIONS ABOUT THE FACE AND UPPER AIR PAS- SAGES *

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The problem of satisfactory anesthesia for operations about the face and upper air passages is worthy of study. The nature of the operations is such that no inhaler or cone can be kept constantly over the face and nose of the patient. It may be necessary to keep the mouth open throughout the operation. Under these conditions it has been impossible by the usual methods to produce a smooth anesthesia.

Fig. 1.—The foot pump, vaporizer and mouth tube connected.

The patients have been anesthetized in the following ways:

1. By nitrous oxid or ethyl chlorid: The anesthesia produced by these agents is too short for the majority of the operations.
2. By charging up the patient with ether and allowing him to recover partially as the operation goes on, then repeating this process as many times as necessary: This method is dangerous and seriously impedes the operation.
3. Chloroform administered with the Junker apparatus—the routine method in England: Chloroform is a dangerous anesthetic for all these operations, and especially in the adenoid cases, in which the patients are

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