ach, that the longer the fast, the less the amount of lactic acid; there being only a trace of it in all instances where the fast was twelve hours or more.

A glance at the report will show that the smallest quantity of fluid obtained at any one sitting, was 12 cubic centimeters, or nearly half an ounce.

Again, it will be noted that after a fast of twelve hours, there was as much as 45 cubic centimeters of fluid found in the stomach, which proves that the stomach always contains some fluid.

Another point is, that after 271 days it is well preserved, showing almost no evidences of chemie deterioration.

A NEW TEST FOR LACTIC ACID IN THE GASTRIC CONTENTS AND A METHOD OF ESTIMATING APPROXIMATELY THE QUANTITY PRESENT.

Presented to the Section on Practice of Medicine, at the Forty-eighth Annual Meeting of the American Medical Association, at Philadelphia, Pa., June 1-4, 1897.

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In view of the value of the detection of lactic acid in the gastric contents, especially in the diagnosis of gastric carcinoma, the importance of which has been increased by the later work of Boas, and because of the uncertainty and unreliability of Uffelmann's test, I present the following.

The test which I detail is simple and easily applied, the reaction is characteristic, the results reliable. It also affords a method of approximately estimating the quantity of lactic acid present. The test solutions used are:

Solution No. 1, consisting of saturated alcoholic solution gentian violet, 0.2 c.c.; distilled water, 500 c.c.

Solution No. 2, solution ferric chlorid (U. S. P., 1890), 5 c.c.; distilled water, 20 c.c.

The method of applying the test is as follows: Into a small porcelain capsule place 1 c.c. solution No. 1 and add from a pipette 1 drop of solution No. 2. The violet color of solution No. 1 changes to a bluish violet after the addition of the ferric chlorid. To this mixture add drop by drop the filtered gastric contents. If lactic acid is present the color of the solution changes from a *bluish violet* to a *green or greenish yellow*.

Alcohol, glucose, butric acid, acetic acid and phosphates, in quantities below 2 per cent. do not interfere with the reaction as they do in Uffelmann's test. The reaction is not interfered with by acetone or albumoses; sulphuric, nitric and hydrochloric acids do not give the reaction.

Delicacy of the test.—One drop of a 0.02 per cent. solution of lactic acid gives a very distinct reaction. The usual limit set down for Uffelmann's test is the detection of 0.05 per cent. If phosphates are present there is at first a reddish violet color produced, which in the course of a second or two gives way to green. If phosphates be present to the extent of 0.5 per cent. it may take two or three drops of a 0.02 per cent. solution of lactic acid to bring out the reaction distinctly.

To determine approximately the quantity of lactic acid present the procedure is as follows: It is well to prepare a 0.2 per cent. solution of lactic acid to use as a standard. Take two narrow test tubes of equal size and to each add 5 c.c. of test solution No. 1 and

5 drops of test solution No. 2. To one tube add 0.5 c.c. of a 0.2 per cent. solution of lactic acid; the mixture turns green and all traces of violet or blue disappear. To the other test tubes add, drop by drop, the filtered gastric contents until the color is the same shade of green as in the first test tube. Comparison may be made more accurate by equalizing the volumes of the solutions in the test tubes by adding to the first tube as much distilled water as was added of gastric contents to the second tube. To get the percentage of lactic acid present, divide 0.1 by the number of cubic centimeters of gastric contents used.

This is not given as a means of determining accurately the amount of lactic acid present, but with care the amount present can be estimated within 0.1 per cent. and to one accustomed to the use of the test a per cent. as low as 0.05 can be estimated.

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THROMBOSIS OF THE JUGULAR VEINS.

Presented in the Section on Practice of Medicine, at the Forty-eighth Annual Meeting of the American Medical Association, held at Philadelphia, June 1-4, 1897.

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When Lasègue in 1879 reported a case of phlebitis of the neck, he stated that after painstaking search, he was unable to find a similar observation published, either in France or elsewhere. Since then a number of cases of this kind have been reported, yet they are sufficiently rare to make each one of peculiar interest.

The following record is that of a patient admitted to the New York Infirmary for Women and Children, in the service of Dr. Josephine Walter, to whom I am indebted for these clinical notes.

Ida D., aged 19 years, American, seamstress. Admitted to hospital Nov. 30, 1896.

Family history. Negative. Mother died from an accident, father and two brothers living and well.

Personal history. Measles when 8 years old. Rheumatic fever when 10 years old, with two subsequent attacks on two succeeding years. Her heart has troubled her from early childhood, beating rapidly whenever she ran or exercised vigorously. This became much worse after the attacks of rheumatism, so that now she has palpitation on every exertion. For several years she has not been strong. During the past year the menses have been irregular, and for the past four months she has had amenorrhea.

Present illness. About five weeks ago patient noticed that her feet became slightly swollen. The swelling, which was painless, has become gradually more marked since then. A few days ago an aching pain developed in the left axilla and in the left side of the neck. There was also swelling in the neck, which when first noticed would disappear when she sat up, and reappear when she lay down.

On admission to the hospital, the patient appears very weak. There are cyanosis and dyspnea present. Temperature 96.2 degrees F. (35.7 degrees C.). Pulse 108, respiration 40.

Examination December 1. There is lividity of the lips with dilatation of the veins on the edge of nostrils and tip of nose. The pupils are widely dilated. The tongue is slightly coated. The pulse is small, rapid and compressible. The dorsum of the left hand is markedly swollen. Both legs are swollen and pit on pressure. There is marked cyanosis of the skin. The glands in the groin are not enlarged. *Heart.* Left border, 4.3 cm. outside nipple line; right border, 2.5 cm. to right of sternum. Double aortic and mitral murmurs. Apparent dilatation near base of heart, which suggests an aneurysm. Very slight murmur heard in the vessels of the neck. This is out of proportion to the sounds at the base of the heart. A systolic murmur is transmitted down the sternum.

On the left side of the neck there is a hard, tender swelling of the lower part of the external jugular vein, which is tortuous. There is a hard lump in the left axilla, extending down the arm for about five inches. The left arm is edematous and quite pale, not cyanosed. The liver is enlarged. There is fluid in the abdominal cavity.

Urinalysis. Amount in twenty-four hours 420 c.c. Turbid, reddish brown in color, reaction markedly acid. Sp. gr. 1033, albumin present. Urea 2.8 per cent.; no sugar. Amorphous urates. A few uric acid crystals. Granular, hyaline and epithelial casts.

December 2. Patient slept very little during the night. Pulse small but regular. There is a slight puffiness in the palm of the hand, but the edema on the dorsum is less marked today. She does not have earache. The external jugular vein is felt as a cord running up the side of the neck to about one-half inch below the lobe of the ear. There is bifurcation of this cord about half way up the neck. Nothing felt passing

the first day it reached 98.2 degrees F. (36.8 degrees C.), but after that it varied between 94 F. (34.4 C.) and 96 degrees F. (35.5 degrees C.). On the fourth day it was discovered that while the temperature was 95 F. (35 C.) by mouth, it reached 101.2 F. (38.4 C.) by rectum, and after this, although there was especial precaution taken to avoid an error in registering, there was a difference of from 6 to 7.8 degrees F. (3.3 to 4.3 C.) between the temperature by mouth and that by rectum. (See temperature chart.)

December 4. Pulse seems stronger and there is less tenderness in the arm and hand. There is slight vibration felt in the first intercostal space near the sternum. In the same position

Thrombosis of internal and external jugular, subclavian and axillary veins. Diagnosis, endocarditis, endophlebitis.

posteriorly. The whole left side of the neck is fuller than the right, following the course of the sterno-cleido-mastoid muscle. A prominence, circumscribed, about two inches long, is found on the inner aspect of the arm just below the axilla. The axilla is not as hard or as tender today, and the feet are less edematous. In the lungs, posteriorly there is dulness in the lower part on both sides, and voice sounds are heard over this region. There are fine crepitant râles heard near the bases. Urine in twenty-four hours only about 135 c.c.

The temperature, which was taken by the mouth during the first five days of her treatment, was persistently low. During

there is slight dulness on percussion, which extends to the second intercostal space, and lies a little further to the left than to the right. The first and second sounds of the heart are heard in the first intercostal space. The character of the pulse is slightly weaker in the left than in the right wrist.

December 5. Slept a good deal during night, but restless during sleep. At one time coughed up a little blood. Dulness in both lungs posteriorly from the angle of the scapula to the base. On auscultation, over the upper part of chest on both sides, the inspiratory murmur is harsh. Fine crepitant râles are heard at the end of inspiration. The heart murmur is not

heard in the back behind the posterior axillary line. Percussion reveals presence of fluid in the thoracic cavity. Thickened cord extending more markedly down side than arm, this morning.

Urine. Amount in twenty-four hours, 510 c.c. Very clear, amber in color, markedly acid. Sp. gr. 1020, albumin 5 grams to liter. Urea 1.5 per cent.; no sugar. Epithelial and hyaline casts.

December 6. Patient failed rapidly, and died in the afternoon.

The postmortem examination was made by Dr. Elizabeth Mercelis. As permission for the section was restricted to that which could be made through the ordinary median incision, the veins of the extremities could not be examined. The record is briefly as follows:

Body moderately nourished. There is general edema, which is most marked in legs and feet, least in upper extremities and chest. On opening the abdominal cavity, there is a free escape of turbid yellow fluid. In both pleural cavities there is also a considerable amount of fluid, with a few firm adhesions between parietal and visceral pleuræ. The apex of heart is in the fifth intercostal space in the mammillary line. In the pericardial sac there is a considerable amount of a clear amber fluid.

The heart is much enlarged, being 16.5 cm. in length, 10 cm. in width and 7.5 cm. in thickness. The right auricle is very greatly distended, measuring 13.5 cm. in length. It is completely uncovered by lung. It presents somewhat the appearance of a great distended vein. The left auricle appears normal. Right ventricle shows lines of fat distinctly marked. On the posterior surface is a considerable area of puckering, as of sclerotic change. The vessels on the surface are distended. The ventricle is filled with fluid blood. Length of cavity 8.5 cm.; thickness of wall 1.6 cm. The tricuspid valve is thickened throughout in ridges, and the edge is curled and thickened. There are no vegetations, and no ulcerations. *Pulmonary valves.* Two of these are fairly thin and flexible; along the free edge of the third (the aortic cusp) there is much thickening, having a firm, almost cartilaginous feel.

Left ventricle. The wall is the same thickness as the thickest part of the right. The endocardium is smooth. The aortic valves are thickened on edges. The aorta is soft and smooth. The mitral valves are very much thickened, and adhesions between the different cusps form an almost uniform piece about the ring. There are no ulcerations or vegetations.

The left innominate, left subclavian, and the external and internal jugular veins, extending up nearly to the lobe of the ear, are filled with firm thrombi. There are ragged bits of thrombus extending down into the superior vena cava.

Behind the left auricle, and pressing upon it, is an enlarged, hard lymph node, 3.7 cm. in length, 2.5 cm. in width and 1.2 cm. in thickness. Extending up each side of the trachea is a chain of enlarged lymph nodes, each about the size of a pea. One of this size is situated at the junction of the subclavian and internal jugular veins. None of these seem to press upon the veins. There is a small extravasation of blood, which has dissected up the left side of the trachea and around the enlarged lymph nodes behind the left auricle. The origin of this blood can not be found.

The lungs are congested throughout and both show, on microscopic examination, an early stage of lobar pneumonia. There are areas of infarction in both lungs.

The kidneys show cloudy swelling, with scattered areas of old connective tissue degeneration.

The liver extends 5 cm. below the margin of the ribs. It is the seat of very extensive fatty change.

The mucous membrane of stomach and intestines is congested.

The spleen is markedly enlarged, congested and friable.

The large veins of abdomen are free from clots.

The uterus and appendages are normal.

On microscopic examination of the thrombosed veins, the internal and external jugulars present the usual picture of organizing thrombi. There are delicate lines of connective tissue extending into the mass of cells which obliterates the lumen of the vessel. The endothelial cells are slightly increased in number, as are the connective tissue cells in the walls of the veins, but these changes are evidently secondary to the thrombus formation. In the subclavian vein, on the other hand, there is an endophlebitis which is evidently of longer duration and primary to the formation of the clot. Around nearly one-fourth of the

circumference the intima is fairly normal, and here the clot lies free in the lumen. The greater part of the intima, however, is much thickened and very irregular along the inner border. In places the lumen is encroached upon to one-fourth or one-third its diameter by this thickened intima. At one portion, the blood clot and intima have become so blended, through the proliferation of intima cells and formation of fibrous bands, extending from the media into the center of clot, that the line of demarcation between them can not be determined. The middle and outer coats are thickened throughout the corresponding portion of the vessel. It appears as if the thrombus formation has been caused by the obstruction to the current of blood caused by this endophlebitis, the stasis being favored by the failure of compensation in the heart.

Bouillard considered that there was a rheumatic phlebitis, similar to rheumatic endocarditis or pericarditis. In this patient's history there is a record of three attacks of acute articular rheumatism, but the last occurred seven years before the present illness.

In 1887, Friedrich Pohl published an article on thrombosis of the subclavian and jugular veins. In this he reported a case, and reviewed the literature of the subject, finding seven cases in which the jugular veins were involved. The records are briefly as follows:

1. Case reported by Pohl, of carcinoma of the vagina, with metastatic growths throughout the viscera. An enlarged carcinomatous lymph node, pressing upon the junction of the subclavian and jugular veins, led to thrombus formation in the left brachial, subclavian and jugular veins, which extended downward for the distance of 1 cm. into the left innominate vein. There were no areas of infarction in the lungs. Death ensued six days after the appearance of the thrombus.

2. An imperfect record of a case reported in 1860, in which the innominate, subclavian and jugular veins were the seat of thrombi caused by the compression of neighboring organs.

3. Ramskill, 1874.—Case of cancer of the uterus, with thrombosis in left external jugular, internal jugular, subclavian and innominate veins and in the superior vena cava. The immediate cause of thrombosis if not given. The patient died one week after the symptoms of thrombus appeared.

4. Walter Smith, 1870.—History of this case not known. The autopsy revealed a large, dark bronchial gland pressing on the superior vena cava, which had caused stasis and thrombus formation in the right internal jugular, the subclavian and axillary and the right innominate veins. There were no metastases, no pyemia or other phlebitis. The heart was dilated, the tricuspid valve thickened, the other valves normal.

5. Cludomiro reported a case of phthisis with cavity formation at the left apex, and adhesions between the pleura and left innominate vein causing stasis and thrombosis in the subclavian, axillary, deep brachial, cephalic, basilic and jugular veins. The patient died fifteen days after the appearance of the clot.

6. Moizard, 1875.—A case of sarcoma of the mediastinum, obliterating the lumen of the superior vena cava, and causing thrombosis of this vessel as well as of the right innominate and right external jugular. The patient died after three or four months.

7. Robert, 1880.—A patient in the later stages of "lung disease," with an enlarged lymph-node, pressing upon and adherent to the left innominate vein, with thrombosis in this vessel and in the internal jugular. The patient died four months after the thrombus formed.

8. More, 1882.—A case of aneurysm of the ascending aorta, with pressure on the superior vena cava, with a large clot occupying the right innominate, subclavian and external jugular veins. Death eight days after the appearance of the thrombus.

Besides these cases reported by Dr. Pohl, I have found records of the following:

9. Lasègue, 1879.—A case of phthisis with thrombosis of the external jugular, subclavian and humeral veins. The thrombus softened and all symptoms of phlebitis disappeared ten days after its formation. No direct cause was discovered in this case.

10. Languebeck, 1881.—A case of tumor of the neck, adherent to, or growing from the internal jugular vein, with thrombosis at that point. Operation with removal of tumor and of thrombosed portion of vein. Recovery. Tumor supposed to be a syphilitic gumma.

11. Pettit, 1882.—A case of yellow fever followed by symptoms of pyemia. At autopsy the right subclavian, external jugular and cephalic veins were occluded and filled with coagulated blood and fibrin. There were areas of infarction and metastatic abscesses in the lungs. Death twelve days after clot formed.

12. Lejard, 1882.—A case of caseous pneumonia with cavity formation. Thrombus, which was supposed to be due to the cachectic condition of the patient, formed in the external jugular and its anastomosis with the anterior jugular, and in the deep veins of the forearm. Death two days after the formation of the clot.

13. George Wilkins, 1883.—Case in which the superior vena cava was converted into a firm, fibrous cord with organized thrombi extending into the innominate and internal jugulars on both sides. The thrombus was thought to be due to an endophlebitis, caused by strain. Death four months after the symptoms of thrombosis in the neck.

14. Kelsch, 1887.—A case of carcinoma of the stomach. Thrombi found in the left external jugular and subclavian, also in the inferior vena cava, from the renal vein to the bifurcation of the vessel, and in the iliac and crural veins on both sides. The patient died one month after the formation of clot in the external jugular vein.

15. Picot, 1884.—Case of phthisis, with the brachio-cephalic trunk of left side obliterated by a clot which extended into the jugular and axillary, but not into the vena cava. This thrombosis was due to compression by a hypertrophied lymph-node.

16. Comby, 1892.—A case showing symptoms of occlusion of the superior vena cava with thrombi in the left external jugular and left axillary veins. The cause was not found. There was a history of syphilis, but no improvement under antisyphilitic treatment. Under treatment with Fowler's solution the patient made a perfect recovery.

17. Younge, 1895.—Case of thrombus formation in superior vena cava, left innominate and jugular veins and left lateral sinus, occurring in Bombay. The condition probably due to exposure to extreme heat, with possibly malarial infection.

18. Gatay, 1896.—A case of rheumatic phlebitis, with death following thrombosis of the subclavian, jugular and innominate veins.

19. Renouard.—Case of rheumatic phlebitis, involving internal jugular and subclavian veins.

20. Nicolle, 1897.—A case of endocarditis, attacking the mitral valve with phlebitis of the external jugular vein.

I was unable to examine the original papers in the last three cases. In all the above cases, the thrombus was found in the jugular vein alone, or in that, together with the larger trunks below the vein.

In the internal jugular there may also be thrombus formation secondary to diseased conditions in the lateral sinus. Indeed, Gatay makes the assertion that phlebitis of the deep jugular vein is regularly secondary to inflammations of the sinus.

In 1880, Lemoine reported a case of death from thrombus formation in the lateral sinus and internal jugular vein in a tuberculous patient, the other veins of the neck being normal.

Allport, in an article on "Purulent brain deposits and phlebitis and thrombosis of the cerebral veins and sinuses following ear disease," found that out of the 169 cases which he reported, in nine the internal jugular was involved. All of these nine died.

Pitt in 1890, in an analysis of fifty-seven cases of ear disease and of the complications which led to death, found that in eleven of these cases the clot extended into the internal jugular, with death from pulmonary pyemia.

Herczel in 1893 reported a case of thrombosis of the transverse sinus and jugular vein, with recovery after operation.

Jansen, in an article published in 1893, on brain thrombus following middle ear disease, found the

internal jugular vein involved in twelve out of 215 cases reported.

In studying the causes of thrombosis of the jugular veins, from an examination of the above records, we find that, as in any situation, the conditions favoring coagulation are: 1, sluggish or arrested circulation; 2, inflammatory processes in the vein due to injury or to bacterial invasion, or to the presence of irritating substances held in solution in the blood; 3, morbid conditions of the blood rendering it more readily coagulable.

Out of the thirty-four cases found, in eight, or nearly a fourth, there was pressure on the vein leading to stasis. In sixteen, or nearly half, there was a grave blood dyscrasia, three having carcinoma, one, sarcoma; six, tuberculosis; two, tertiary syphilis; one, pyemia following yellow fever; two, acute articular rheumatism, and one probably malaria combined with changes due to heat exposure. In five cases there is endophlebitis, three probably due to rheumatism, one to syphilis and one to injury. In thirteen cases, or 38 per cent., the condition is secondary to middle ear disease.

In only one of these cases, that of syphilitic gumma, does the morbid process seem to have originated in the jugular vein itself. In the others it formed secondary to thrombus in the innominate vein, extending away from the heart, or secondary to clot in the lateral sinus, extending toward the heart. In many of the reports it is stated that the clot was adherent to the vessel wall, showing that there had been an endophlebitis, either secondary to the thrombus formation, or primary and leading to it.

The symptoms are those of phlebitis in any vein. Following the description of Gatay, these are pain, tenderness, edema, venous arborization, the presence of an indurated cord, and a line of redness if the vein be superficial. In one case the edema was limited by the median line of the neck. In that of Dr. Walter, there was a peculiar subnormal temperature by mouth, while that by rectum was elevated. The general symptoms are usually masked by those of the original disease.

While the prognosis of venous thrombosis is, in general, not very grave, it would seem from a study of the above cases, that in the jugular veins it is seldom found excepting in the last stages of fatal diseases. In sinus thrombosis, the involvement of the jugular vein makes the prognosis much more grave from the liability to pulmonary infarction. In only four of the cases noted above was there recovery. One, occurring in the course of phthisis, made a spontaneous recovery; one, of doubtful etiology, recovered under the use of Fowler's solution; one of syphilitic tumor recovered after operation, as did one of sinus thrombosis following ear disease.

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