lactating cows has been demonstrated by Forbes and associates.⁶

The total blood of a 60 kg. man contains about 0.3 gm. of calcium. The bones contain approximately 4,000 times as much. With this great reserve supply of calcium, a portion of which is available, as is indicated by the great constancy of blood calcium under varying diets and conditions, a physiologic basis for the administration of calcium salts in tuberculosis has yet to be demonstrated.

SUMMARY AND CONCLUSIONS

Determination of the calcium content of the blood of patients with advancing and convalescing tuberculosis revealed that in the incipient cases in which the patients, who were on a high milk diet, showed marked improvement, the values for calcium in the serum were normal and fairly constant. In no case was the calcium value increased above normal figures by this high calcium diet. In advanced cases the variations obtained were greater (some rather high and some rather low values being obtained), and improving patients showed on the average slightly higher values than the unimproved. No marked deviations from the normal, however, were observed in the calcium content of the serum of patients in the various stages of pulmonary tuberculosis.

The failure of the body to deposit lime around the tuberculous areas is to be ascribed, not to a deficiency in blood calcium, but rather to an inability of the cells of the tuberculous area properly to utilize available calcium.

ICTERUS GRAVIS SYPHILITICUS

ITS RELATION TO ACUTE YELLOW ATROPHY

UDO J. WILE, A.B., M.D.

Professor of Dermatology and Syphilology, University of
Michigan Medical School

AND

ROLLA G. KARSHNER, B.S.

ANN ARBOR, MICH.

Jaundice, occurring as a rare complication in the onset or early secondary period of syphilis, was first recognized by Paracelsus in 1585. It is also mentioned by Portal, Massa, Monti, Batallus, Fallopio and other writers of the latter part of the sixteenth century, in accordance with an early belief that the liver was the site of election for venereal disease. At a later date Ricord¹ recorded icterus in two cases of early syphilis and ventured the statement that syphilis was the cause of the jaundice. In 1853 Gubler² substantiated the work of Ricord with a report of seven cases, three of which he collected from the literature, and by clinical observation proved the jaundice to be syphilitic. Subsequently Lancereaux³ collected reports of twenty cases of syphilitic icterus.

Jaundice in early syphilis is now recognized as occurring in two forms, mild jaundice and severe jaundice, or icterus gravis. The mild form, which is by far the more common, differs very little in symptomatology from ordinary catarrhal jaundice. From an

analysis of 15,799 cases of early syphilis in Engel-Reimers' clinic, Werner⁴ found that fifty-seven, or 0.37 per cent., were jaundiced. Engel-Reimers⁵ reports eleven cases of icterus occurring among 759 cases of early syphilis in women, or 1.4 per cent. The difference in these figures represents fairly accurately the more common occurrence of the condition in women. In Engel-Reimers' series, 80 per cent. of the patients developed the jaundice within three months after infection. In Michael's⁶ analysis of twenty-five cases, in three the icterus appeared before the chancre, in one case with the chancre. While most common at the time of the exanthem, a small percentage of the cases occur late in the secondary period, and not coincident with the eruption. In general, the onset is more sudden than in catarrhal jaundice, and there is often an absence of gastro-intestinal symptoms, though too much stress must not be laid on this point, as gastro-intestinal symptoms often accompany the florid stage of syphilis. The appetite is generally good, though there may be a distaste for fats. In some cases the stools are clay colored; in others they are normal. Swelling of the liver is a fairly uniform finding. The spleen is often enlarged, but this may be attributed to the generalized spirochetosis of the secondary period.

The icterus extends over a few weeks and disappears, particularly under treatment, though cases are reported in which it has become chronic. In twenty-five cases collected in seven years from Buschke's⁷ clinic, the duration was never shorter under treatment than two weeks, with an average of four weeks. In many instances it has developed into the grave form. That it is not due to the administration of mercury is proved by the fact that it often appears when mercury has not been administered, and readily disappears on intense mercurialization.

The three cardinal points brought out by Gubler, the absence of other cause for the jaundice, its usual appearance with the general eruption, and the rapid disappearance on specific treatment, are the features which still serve clinically to differentiate the syphilitic condition from simple catarrhal jaundice. Salomon⁸ regards early syphilitic jaundice as an indication of feeble resistance, and therefore of prognostic importance. Four of Richter's⁹ forty-one collected cases were malignant syphilis.

The explanations of the cause of the mild form of syphilitic jaundice are many and varied. Gubler believed it to be due to a roseola or exanthem of the intestine and bile duct. French writers in general regard it as the result of swelling of the portal lymph vessels. Engel-Reimers⁵ was able to show at necropsy in three cases that it was an obstructive jaundice caused by syphilitic adenitis of the portal lymph glands. Lubinoff¹⁰ has reported the necropsy of a case of syphilitic icterus associated with precocious late manifestations in which the ductus choledochus was occluded by pressure of an enlarged lymph gland. Senator¹¹ holds that the jaundice is due to an inflammatory reaction. Neumann¹² says that all of the fore-

4. Werner: München. med. Wehnschr., 1897, **44**, 736.

5. Engel-Reimers, Julius: Monatsh. f. prakt. Dermat., 1892, **15**, 475.

6. Michael: Arch. f. Dermat. u. Syph., 1914, **120**, 694.

7. Buschke: Berl. klin. Wehnschr., 1910, **47**, 238.

8. Salomon, cited in Loomis, A. L., and Thompson, W. G.: System of Practical Medicine, by American Authors, New York, Lea Brothers & Co., 1898, **3**.

9. Richter: Charité-Ann., 1898, **23**, 365.

10. Lubinoff: Méd. mod., 1894, No. 1, p. 13.

11. Senator: Charité-Ann., 1893, **18**, 322; *ibid.*, 1898, **23**.

12. Neumann: Spezielle Pathologie und Therapie, Vienna, 1899, **23**, 449.

6. Forbes and Beegle: Bull. 295, Ohio Exp. Sta., 1916.

1. Ricord: Clinique iconographique de l'Hôpital des vénériennes, 1851.

2. Gubler: Compt. rend. Soc. de biol., 1853, **5**, 255; Gaz. méd. de Paris, 1854, No. 13, p. 186; *ibid.*, No. 15, p. 214, etc.

3. Lancereaux: Traité historique et pratique de la syphilis, Ed. 2, 1866, p. 146; Traité de la syphilis, Ed. 2, Paris, 1873.

going factors may be coincident. In view of the swelling of the liver, Mauriac¹³ believes that the jaundice is due to an active hepatitis with hyperemia and stopping up of the biliary ducts. Finger¹⁴ says that it is due to syphilotoxin. This is essentially the explanation offered by Fournier.¹⁵ Still others ascribe the cause to a fear of the disease which manifests itself on the appearance of the secondary lesions. Talamon¹⁶ has described an interesting case in a girl aged 17 in whom such a fear brought about an "emotional jaundice" within eighteen hours, followed in three weeks by death from acute yellow atrophy. He states that the emotional jaundice probably renders the liver more susceptible to the effects of the syphilotoxin.

Of far greater importance than mild icterus is the grave form, which often supervenes on the former, though it may arise insidiously with a stormy onset and rapid course. The tendency if untreated is for the condition to go on to acute yellow atrophy. In 1854 Lebert,¹⁷ out of a collection of seventy-two cases of acute yellow atrophy, reported seven as associated with early syphilis. Tierfelder¹⁸ found that eight out of eighty-one cases of acute yellow atrophy occurred in old or recent syphilitics. In Osler's *Modern Medicine*,¹⁹ it is stated that 10 per cent. of all cases of acute yellow atrophy occur during secondary syphilis. Richter,⁹ in 1898, collected reports of thirty-nine cases of syphilitic icterus gravis from the literature, and added two of his own. Umber²⁰ collected reports of fifty cases. In Michael's⁶ analysis of forty cases of jaundice in early syphilis, four, or 10 per cent., went on to acute yellow atrophy. The condition seems to be much more common in women, the ratio being about 5:1. In 1894, Lasch²¹ named the condition "icterus syphiliticus praecox," and many cases were subsequently reported under that title. In fifty cases reviewed by him, mercury had been administered in only three previous to the appearance of the icterus. Of thirty cases, the icterus occurred in twenty-five within three months after infection. In most of the cases reported in the literature, the icterus has occurred coincident with the exanthem. It may appear earlier. Practically all cases begin sometime during the first year of infection. Bendig²² has recently observed a case similar to the one herein reported, in which the icterus occurred subsequent to the complete disappearance of the secondary syphilitic lesions under mercury rubs. He argues that it is not sufficient to cure the manifest lesions of the syphilis, for when these have disappeared, there is within the body, still present, the syphilotoxin.

The cause of grave icterus was carefully studied by Buschke and Zernik,²³ in 1911, who concluded that it was due to a syphilotoxic parenchymatous hepatitis, and not an obstructive jaundice. This view was earlier put forth by Buraczinski,²⁴ and Vespremi and Kanitz,²⁵ and at present is generally accepted as cor-

rect. Other factors may sometimes play a rôle. Engel-Reimers⁵ and others report cases in which pregnancy complicated the condition. Erichson's¹⁸ case followed abortion. In Richter's⁹ forty-one cases, a few were complicated with gonorrhea, others with sepsis. None had previous liver disease. Mercury does not play a part in the causation. Jaundice has been noted as occurring in the course of salvarsan injections, and a salvarsan icterus, often leading to death, is reported by Milian,²⁶ Rille,²⁷ Pinkus,²⁷ Klausner,²⁸ Arno Hoffmann,²⁹ Levy, Bing and Duroeux,²⁷ Dujardin³⁰ and Lube.³¹ Milian,²⁶ who has studied these cases at length and reported several of them, has come to the conclusion that they are in no way due to the salvarsan itself, but that they are in the nature of hepaticorecides, analogous to those occurring in the nervous system. In each case there results a lighting up of a syphilitic process in the liver and a resulting hepatitis which calls for more salvarsan, just as in the case with recurrences in the nervous system. The case of grave icterus which is described herein followed salvarsanization, but could not in any way have been due to the salvarsan, as it appeared abruptly four months after the last salvarsan injection had been given.

The symptomatology of icterus gravis is that of acute yellow atrophy, differing chiefly in the fact that it yields to specific treatment when recognized early. The onset is variable, generally insidious. A mild icterus usually appears as the first objective sign, occasionally accompanied by malaise, vomiting and constipation, but usually without gastro-intestinal symptoms. There may be a slight rise in temperature, though subnormal temperatures are often reported. The pulse is slightly accelerated. Severe abdominal pain may be complained of. Lumbago and muscular pain are common. There is often a debility similar to that of typhoid fever. This may go on to collapse. Cases are reported with ascites. While not a uniform finding, hemorrhagic symptoms are mentioned by many authors. These are manifested by gingivitis, bloody vomitus, melena, purpuric spots in the skin, and more rarely hemoptysis, hematuria and metrorrhagia. There is often a hemorrhagic nephritis. Pregnant women may abort, but this is not always the case.

The second stage is very definite in onset and is marked by a pronounced change for the worse in the general condition, by rapid decrease in size of the liver, the occurrence of leucin and tyrosin in the urine, and especially by the appearance of nervous symptoms. Headache appears which, if present before, becomes very intense. There may be pupillary disturbances and amblyopia. Mentality is affected. The patient may become depressed, or restlessness may supervene during which there are insomnia, pain, convulsions and delirium. The patient often screams and becomes very violent. Apart from the jaundice, the clinical symptoms may closely resemble meningitis. Partial paralysis, such as squint, has been noted. In Fournier's¹⁵ case there was hemiplegia. Dyspnea may resemble the air hunger of Kussmaul. Constipation extends throughout the condition, and the dejecta are offensive. The patient finally dies in coma with complete relaxation of the sphincters.

13. Mauriac: *Gaz. hebdomadaire de médecine*, 1888, **25**, 564.
14. Finger: *Die Syphilis und die venerischen Krankheiten*, 1892.
15. Fournier: *Traité de la syphilis*, Paris, J. Rueff, 1906; *Leçons sur la syphilis chez la femme*, Paris, 1883, p. 926.
16. Talamon: *Méd. mod.*, Feb. 13, 1897, p. 97.
17. Lebert: *Virchows Arch. f. path. Anat.*, 1854, **7**, 385.
18. Cited by Richter (Footnote 9).
19. Osler and McCrae: *Modern Medicine*, Philadelphia, Lea & Febiger, 3.
20. Umber: *München. med. Wehnschr.*, 1911, **58**, 2499.
21. Lasch, O.: *Berl. klin. Wehnschr.*, 1894, **31**, 906.
22. Bendig: *Berl. klin. Wehnschr.*, 1908, **45**, 1229; *München. med. Wehnschr.*, 1915, **62**, 1144.
23. Buschke and Zernik: *Arch. f. Dermat. u. Syph.*, 1911, **106**, 121.
24. Buraczinski: *Wienn. klin. Rundschau*, 1907, **21**, 651, 699.
25. Vespremi and Kanitz: *Arch. f. Dermat. u. Syph.*, 1907, **88**, 33.

26. Milian, M. G.: *Bull. de la Soc. franç. de dermat. et de syph.*, No. 7, July, 1914, to December, 1915; *Ann. d. mal. vén.*, October, 1911.
27. Cited by Milian (Footnote 26).
28. Klausner: *München. med. Wehnschr.*, 1911, **58**, 570.
29. Hoffmann, Arno: *München. med. Wehnschr.*, 1911, **58**, 570.
30. Dujardin: *Jour. méd. de Bruxelles*, April 12, 1914, p. 182.
31. Lube: *Deutsch. med. Wehnschr.*, 1914, **40**, 946.

The liver is probably enlarged in the onset of all cases, though this is not always easily made out. The enlargement is smooth, the right lobe being most affected. In one third of the cases the liver is tender. After a variable period, reduction in size comes on suddenly with the severe symptoms, and heralds a rapid fatal outcome.

At necropsy the liver presents the typical picture of acute yellow atrophy, often being reduced to one-third its normal size. The more slowly progressing cases show areas of acute yellow atrophy interspersed among areas of red atrophy, the latter representing earlier destruction. In some cases the entire liver is not affected, while in others not a single normal cell remains. Buraczinski,²⁴ Vespremi and Kanitz,²⁵ Fischer³² and Bendig²² failed to demonstrate spirochetes in the liver of cases at necropsy. Buschke and Zernik²³ injected the liver tissue into monkeys with negative results. The spleen is often enlarged.

The urine is diminished in amount and highly colored with bile pigment. There is no glycosuria. Small amounts of albumin often occur together with casts. Albumosuria is reported, and is supposed by some to come from direct destruction of the liver cells. Crystals of leucin and tyrosin are invariably present in greater or less amounts, often in so great an abundance that they spontaneously precipitate, but Rolleston³³ does not regard these as pathognomonic. On the other hand, Senator¹¹ Buschke and Zernik,²³ and others look on their appearance as evidence of beginning liver atrophy.

In some cases the stools contain bile pigment; in others they are clay colored.

There is no characteristic blood picture.

Unrecognized cases are invariably fatal. According to Richter,⁹ the course is somewhat longer than that of acute yellow atrophy from other cause, the average duration being eighty-four days. Many cases are on record, however, as having ended fatally in less than two weeks. Precipitous cases are not uncommon. In one of Bendig's²² reports a rather mild icterus with indefinite prodromal symptoms extended over three days, followed by a stormy exacerbation and death within twenty-four hours. With early specific treatment, the course may be arrested or retarded.

The older writers regarded the condition as always fatal. The prognosis, however, seems fairly good for recovery if the condition is recognized early and intensive specific treatment is begun. Isolated cures of advanced cases by salvarsan are recorded (Duhot,³⁴ Umber²⁰).³⁵ Several cases of recovery have been

reported (Senator,¹¹ Buschke and Zernik,²³ Lasch²¹ and others) even after appreciable decrease in the size of the liver and the appearance of leucin and tyrosin in the urine. The condition should be diagnosed in the stage of enlargement. Purgation with calomel and intense mercurialization, either by injections or rubs, is indicated by most authorities.

During the past year a case of icterus gravis, occurring early during the course of syphilis and resulting in recovery, was under observation in the department of dermatology and syphilology at the university hospital.

The patient, a man, aged 38, married, salesman, entered the hospital for the first time, Dec. 11, 1915. At that time he had a typical chancre of the penis and a maculopapular rash over the entire body. He had been married fourteen years and was the father of four healthy living children. His wife had had no miscarriages. The patient had an attack of gonorrhea at the age of 17. Otherwise the past history was negative.

A sore had appeared on the penis in October, 1915, following a clandestine exposure about six weeks previously. The sore was painless and attended by slight discharge. It had been treated locally by the family physician. The patient had not noticed any glandular swelling. The eruption over the body had appeared one week previous to his entrance to the hospital. He had had slight headache for a few days, distinctly worse at night, and complained that he was not feeling as well as usual, although not definitely ill. He had received up to this time no antisiphilic treatment.

The patient was found to be well nourished with no apparent loss of weight. There were numerous syphilitic papules on the scalp. On the forehead, over the body and on the upper portion of the extremities there was a diffuse maculopapular rash typically roseolous in character. On the right side of the penis at the corona was a typical chancre about the size of a dime with lamellar induration. On the right side of the tip of the tongue there was an involuting mucous patch. The superficial lymph glands were all enlarged. The superficial reflexes were very active and not markedly exaggerated. The spleen was not palpable. The blood Wassermann test was + + + +.

Ophthalmologic examination revealed marked edema and hyperemia of the retina—a low grade neuroretinitis, specific in type.

The neurologic examination revealed slight tendency to stagger with the eyes closed, and pupils which were somewhat sluggish. The lumbar puncture, however, revealed an entirely negative finding: 4 cells, albumin negative, Nonne-Apelt negative, spinal fluid Wassermann test negative. The department of otology reported syphilitic involvement of the eighth nerve.

December 14, the patient received an intravenous injection of 0.3 gm. of arsenobenzol, after which he suffered a marked Herxheimer reaction, the temperature rising to 102.8. This, however, was rapidly reduced on the following day. By December 17, the eruption over the body had disappeared and the primary sore was rapidly involuting.

A second and third injection of arsenobenzol, consisting of 0.4 and 0.6 gm., were given, December 20 and 27, respectively, from which the patient showed no reaction.

The patient said he felt excellent, December 28; the rash had entirely disappeared, and he was discharged from the hospital with instructions to continue his treatment in the form of mercurial inunctions.

April 22, 1916, the patient stated in a letter that he was not feeling well; that he tired easily; that his appetite was failing. He had lost considerable weight and had noticed that his eyes and complexion were becoming rapidly yellow. He was advised to return to the hospital for further observation, and appeared, May 2.

At this time the patient looked very ill. He was intensely jaundiced. The liver was markedly enlarged, extending about 4 finger breadths below the costal margin. The enlargement was smooth and fairly firm, the free border of the liver being rounded. The liver was tender to palpation.

32. Fischer: Berl. klin. Wehnschr., 1908, 45, 905.
33. Rolleston: Diseases of the Liver, Gall Bladder and Bile Ducts, Philadelphia, W. B. Saunders Company, 1905.

34. Duhot: München. med. Wehnschr., 1911, 58, 2113.

35. In addition to the references already given, the following will be found of interest:

Arnheim: Morf. f. prakt. Dermat., 1898, 26, 436.
Brallet: Gaz. d. hôp., 1914, 87, 1063.
Braunschweig: Med. Klin., 1911, No. 4, p. 137.
Brodfeld, E.: Med. Klin., 1912, No. 43, p. 1742.
Castaing: Bull. de l'Acad. de méd., 1913, Series 3, 70, 527.
Chaufard: Presse méd., Aug. 11, 1897, p. 69.
Fagge: Tr. Path. Soc., 18, 136.
Goldstein: Wien. med. Wehnschr., 1904, 54, 1862, 1918.
Hutchinson: Med. Times, 1876, p. 136.
Joseph: Arch. f. Dermat. u. Syph., 1894, 29, 383.
Liebermeister: Deutsch. med. Wehnschr., 1893, 19, 365.
Marchand: Beitr. z. path. Anat. u. z. allg. Path. (Ziegler's), 1895, 17, 206.
Meder: Beitr. z. path. Anat. u. z. allg. Path. (Ziegler's), 1895, 17, 173.
Melchior: München. med. Wehnschr., 1907, 54, 2135.
Moxter, cited by Richter (Footnote 9).
Rosenberg: Deutsch. med. Wehnschr., 1912, 38, 756.
Samberger: Arch. f. Dermat. u. Syph., 1903, 67, 89.
Sevinoff: Thèse de Paris, 1913, Jouve & Cie.
Stroebe: Beitr. z. path. Anat. u. z. allg. Path. (Ziegler's), 1897, 21, 378.
Thurnwald: Wien. med. Wehnschr., 1901, 51, 1379.

The spleen was markedly enlarged and firm, extending about 3 finger breadths below the costal margin. It was not tender. The stools were clay colored, the urine markedly tinged with bile pigment. There was a moderate degree of albumin and there were large numbers of granular casts. The temperature was 98.6, the pulse 100.

The patient was at once referred to the department of internal medicine, and during the course of his stay in the hospital was repeatedly seen in consultation with Professor Hewlett, the head of that department. The abrupt onset without gastro-intestinal symptoms, the enlargement of the spleen, the absence of fever, the intensity of the jaundice and the rapid increase in the size of the liver all pointed to icterus gravis occurring in the early months of syphilitic infection, rather than to a simple catarrhal jaundice. Added weight to this diagnosis was given by the associated nephritis.

The patient was placed under observation for six days, during which time no treatment was given. During this period he became progressively worse, was losing weight at the rate of a pound and a half a day, and a marked decrease in the size of the liver was noted from day to day. Calomel injections, $1\frac{1}{2}$ grains intramuscularly, were now instituted with daily saline catharsis. The patient seemed somewhat improved after the first injection, the most marked effect of the mercury being a diminution in the amount of albumin and the number of casts in the urine. By May 15, the albumin had entirely disappeared from the urine, the stools had become bile pigmented and the jaundice was rapidly clearing up. From this time on, inunctions of 6 gm. of mercury daily were instituted to replace the calomel injections. May 18, the patient was given potassium iodid in increasing doses, beginning with 35 grains three times a day. The liver was still somewhat enlarged over the normal, but was no longer tender.

By May 26, the patient was discharged. At this time he was but slightly jaundiced; the icterus was clearing up rapidly; the liver was no longer palpable over the costal margin. The spleen had also receded to normal in size. He felt well. The urinary findings had entirely cleared up.

The patient reentered the hospital, Dec. 4, 1916, with a gumma involving the skin of the face and the submaxillary gland. The liver at this time was normal to percussion and palpation; there was no trace of jaundice. The examination of the urine was negative and the spleen was no longer palpable. Since his discharge from the hospital, and up to two weeks ago, when the gumma appeared on his face, the patient had enjoyed good health.

CHRONIC NEPHRITIS SIMULATING THE SYMPTOMS OF CEREBRAL NEOPLASM

JOSEPH COLLINS, MD.

Physician to the Neurological Institute
NEW YORK

The symptoms of cerebral neoplasm may be so closely paralleled by other diseases as to make the differentiation very difficult until opportunity has been had for protracted observation and study. Chronic nephritis, for example, may cause all the symptoms of brain tumor. I have seen two such cases recently, and one of them presented so much difficulty of diagnosis that it seems to me desirable to record it.

The patient was an Irishman, aged 57, married, employed as a watchman. The symptoms for which he sought relief had existed about a year. The initial symptom was headache; the pain was principally in the left temporofrontal region. He attributed the pain to an injury which he experienced three years before when he had been knocked down by a bicycle, and had received a wound over the left eye which required nine stitches. Since that time there had always been sensitiveness over the left eye. When the headache began, it would last a few days, and then a considerable period of respite would follow. It gradually grew more

severe, and more continuous, and now he is rarely free from it.

The second symptom to develop was vomiting. It began soon after the headache became severe. The vomiting attacks averaged perhaps once a week at first, and more when the headache became very severe. The third symptom to develop was disorder of vision. For years he had experienced occasional diplopia, to which he gave no attention. It is not apparent on what it was dependent. Six months ago when the vision began to fail he consulted a physician who made the diagnosis of beginning optic neuritis. Meanwhile he began to complain of some general weakness, and during the last two months there has been complete loss of copulative capacity. For the past ten years he has had slowness of urination. He rises once or twice at night to void urine.

Latterly, when the headache is very severe, he has felt giddy. There was no history of convulsion or of paralysis.

His symptoms, in brief, were headache, giddiness, occasional unsteadiness in standing and walking, nausea, vomiting, failing vision and general weakness.

The patient was plump, pale and anemic looking, and the facies was suggestive of nephritis cachexia. The pulse rate was 100, the systolic blood pressure 210, the diastolic 165. The arteries were not particularly thickened or rigid on palpation. The heart was enlarged, and its sounds characteristic of chronic vascular hypertonus, but there were no murmurs. The patient had many bad teeth, all bathed in pus, and roentgenoscopy revealed areas of apparent softening around many of them. The third molar on the left side extended into the antrum.

Examination revealed the motor system normal, except that the right lower extremity seemed somewhat weaker than the left, and there was distinct tremor of the extended fingers. The right knee jerk was slightly livelier than the left; the ankle jerks could not be obtained. The plantar jerks were flexor in type and there was no Babinski phenomenon. The abdominal reflexes were present and equal. The epigastriacs were sluggish. The arm jerks were quite normal, and there was no digital reflex.

Examination of the eyes revealed slight irregularity of the pupils, the right being 4.5 mm. in diameter, the left 4 mm. They were circular, the reaction to light slightly sluggish, that to convergence good. There existed a considerable degree of ptosis, the result of old trachoma, but otherwise the mobility was normal. Examination of the fundi showed pronounced papilledema. It could not be accurately measured. The right disk was elevated to at least 5 diopters, the left about 3. Vision in the right eye with a plus 3 D. was brought up to $\frac{19}{200}$ and in the left eye to $\frac{20}{70}$. There was slight opacity of the right cornea, the result of old trachoma. The fields were complete.

Hearing in the left ear was not as keen as in the right. Examination of the left ear showed a mild chronic sclerotic thickening of the middle ear. The internal auditory apparatus was quite normal. The functions of each of the other cranial nerves were unimpaired. There was no disturbance of speech, and the various forms of superficial and deep sensibility were quite normal.

Examination of the blood serum and of the cerebrospinal fluid gave a negative Wassermann reaction, and there were no cells in the latter, but there was an excess of globulin. The fluid escaped through the needle under considerable pressure. Examination of the blood revealed a typical picture of a secondary anemia: the red blood corpuscles 4,936,000, hemoglobin 88 per cent., white blood corpuscles 6,200, polymorphonuclears 75 per cent., transitionals 4 per cent., large lymphocytes 18 per cent., small lymphocytes 2 per cent., and eosinophils 1 per cent.

Sugar tolerance was quite normal. The estimation of non-protein nitrogen when the patient came into the hospital was 61,100 c.c.; two weeks later it was 52,100 c.c. The urine contained albumin and a few hyaline and granular casts.

During his stay in the hospital the most conspicuous symptom, in addition to the headache, vomiting and choked disks, was vascular hypertonus. The blood pressure fluctuated between 200 and 270, the systolic averaging about 230. A few days after entering the hospital he had an elevation of