denum shows two or three miliary or slightly larger yellowish nodules. Bile papilla patent.

Pancreas.—The region of the pancreas is occupied by a grumous greenish-gray to black mass, in which the only recognizable element consists of fibrous strands, presumably remnants of interlobular pancreatic tissue. The mass is throughout retroperitoneal and has not burst into lesser peritoneal cavity, although rupture is readily brought about by postmortem manipulation. In a few places there are collections of more fluid character. Smears from various regions show no organisms or recognizable cellular elements. The lower portion of the head of the pancreas appears normal. Wirsung's duct can be demonstrated, and contains no stones. This transverse necrotic mass is continuous inferiorly and to the left with similar, perhaps more hemorrhagic material which surrounds the kidney, approaches the bottom of the operative wound and has infiltrated the left retroperitoneal region as far as the left iliac vein and the muscular mass superior to the brim of the pelvis. The surface of lesser peritoneal cavity anterior to necrotic mass shows several slightly raised yellowish nodules from $\frac{1}{4}$ to $\frac{1}{2}$ cm. in diameter, usually several centimeters apart. The relatively normal pancreas at the head shows similar spherules of necrosis. The inferior surface of liver, the superior surface of the mesogaster and the inner surface of the spleen are walled off from the necrotic mass by nothing more than a pale, thin layer of delicate fibrous tissue. The splenic flexure of the colon is not involved in the necrotic process, and only the most posterior portion of its mesentery has been infiltrated.

Liver (weight 1,700 gms.).—Moderately firm and gray-red with hint of yellow, with lighter areas in the reddish mottled field; the centers of pale areas sometimes show slight foramina—the central veins.

Gall bladder.—Pale from thin investment of fibrous tissue. Its ducts are patent, the common duct somewhat dilated. Two mulberry stones of a grayish-yellow color, somewhat smaller than cherries, are found in the gall bladder. No stone or sign of stones in either duct. Bile thick, yellowish green.

Kidneys (weight 365 gms.).—Left kidney: capsule strips with some difficulty, leaving a gray-red surface with red points corresponding to glomeruli. On section the markings can be made out but are indistinct, and the cortex shows a few miliary, irregularly scattered paler areas supporting fat or cloudy swelling. Fresh examination shows very slight intra-epithelial fat. Pelvis smooth, pale, without abnormal contents. Right kidney: Capsule strips readily, markings distinct. Kidney as a whole looks redder than the left. No intra-epithelial fat. Pelvis normal.

Adrenals.—Left adrenal necrosed and unrecognizable. Right adrenal normal. Bladder and genitalia normal.

Aorta.—Shows transverse atheroma above sinuses of Valsalva, and a few narrow, slightly raised yellowish areas down the arch parallel with major axis.

ANATOMICAL DIAGNOSES.

Necrosis of body and chief portion of head of pancreas.

Necrosis of left adrenal.

Necrotic mass in entire left retroperitoneal (perinephric) region, but not involving renal substance. Fat necrosis of the peritoneum.

Hepatic congestion.

Mitral atheroma.

Aortic atheroma.

Chronic pericarditis (milk patches).

Chronic adhesive pleuritis, bilateral.

Operation wound.

Localized peritonitis opposite wound.

Cultures, negative.

MICROSCOPICAL EXAMINATION.

Liver.—A fairly coarse vacuolation of varying diameter but in many cases as broad as a cell-cord, prefers the outer third of the lobule. The epithelium is everywhere granular. The interlobular tissue surrounding certain capillaries, and a few focal areas where the fatty transformation is at its height, show a good many polynuclear leucocytes. In places vacuoles in cells contain hyaline globules.

Lung.—Bronchi well preserved. Peribronchial anthracosis.

Kidney.—Epithelium swollen and granular.

Heart.—Pigment in moderate amount.

Pancreas.—Portion from normal-seeming head of pancreas (*v. gross* description) shows some fairly normal lobules, between which are comparatively wide reaches of interlobular tissue, into which masses of blood have been effused. In places the connective tissue nuclei remain or may be reinforced by lymphoid nuclei; but elsewhere the interlobular tissue may be represented by granular eosinophilic debris, with linear spaces once occupied by crystals. In one place fat tissue is well preserved.

Spleen.—Trabeculae and Malpighian bodies normal. The chief constituents of the pulp are evenly scattered lymphoid cells and blood globules. Plasma cells are prominent; and almost as many polynuclear leucocytes appear on closer search.

REMARKS.

Attention is especially directed to the following points:

(1) The previous existence of two attacks, presumably due to gall-stones, the connection of which with a subsequent pancreatitis is rendered probable by constantly accumulating evidence.

(2) The extension of the fat necrosis towards the left adrenal and kidney, emphasizing the value of posterior drainage in such cases.

(3) The possible importance of the destruction of the adrenal in producing the fatal issue.

(4) The negative results of bacteriological examination in extensive necrosis of the pancreas and fat tissue.

CEREBRAL SYPHILIS.¹

BY ALBERT E. BROWNRIGG, M.D.,

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THAT syphilis may cause severe disturbance of the internal organs is a comparatively modern idea. Most of the earlier writers ascribed the associated nervous symptoms rather to the effects of treatment than to specific organic changes. They devoted their whole attention to describing the external ap-

¹ Read before the Nashua, N. H., Medical Society.

pearances of the disease, though there were some who mention the occurrence of "internal pustules" and of the rising of "syphilitic vapors" to the brain.

Ballonius, in the early part of the seventeenth century, was the first to definitely describe syphilitic gumma of the brain. But postmortem examinations were rare, and so it is not surprising to find that Hunter in 1787 but expressed the prevailing opinion of his time when he taught that the internal organs and especially the brain could not be affected with syphilis.

Schutzemberger, in 1850, published the results of his studies on cerebral syphilis, and directed general attention to this subject, which Virchow and other pathologists soon elaborated with their accumulated data from the postmortem table. So fascinating and prolific did this topic become, that a host of investigators made it their special field, and within the last decades it has been brought from its former obscurity to one of the most clearly recognized and definite of brain affections.

ETIOLOGY.

Why syphilis should attack the central nervous system in some and not in others similarly infected has caused much discussion.

Age, sex, environment and nutrition would seem not to be determining factors in predisposing to special lesions of the nervous system. It used to be thought that the use of mercury tended to inflammation and degeneration of the brain, and that consequently those cases that were vigorously treated with mercurials were more apt to have syphilitic brain disease. This theory was not well borne out by clinical facts and, besides, Kussmaul and others have clearly proved that the degeneration from mercury is of an entirely different anatomical character.

Virchow first called attention to the undoubted fact that the localization of syphilitic lesions in the skin, bones, etc., is often determined by external injuries and accidents. If this is true of the tissues in general, it would seem to afford a reason why those cases in which the nervous system is subjected to excessive work, worry, shock, or irritation from intoxicants are the ones most likely to have cerebral lesions supervene. For similar reasons, we can understand that persons inheriting a neurotic constitution would thus afford a vulnerable point to the inroads of the poisonous principle, and it is a clinical fact that such persons are particularly liable to have lesions develop in the nervous system comparatively early in the disease.

PATHOLOGY.

To intelligently comprehend the variety but essential unity of the cerebral lesions of syphilis, it must be borne in mind that they are all essentially inflammatory in character. It is generally conceded that syphilis is due to infection with a specific microbe, and the result of its growth in the nervous system, as elsewhere, is a peculiar form of exudative inflammation. The chief seat of its activity is in the walls of the cerebral blood vessels, with consequent arteritis and impairment of nutrition of the brain tissue beyond the lesion. This leads to localized edemas and ischemias, areas of softening and manifold impairment of function.

Associated with this inflammatory reaction there exists a peculiar exudation or infiltration about the affected vessels. These infiltrated areas are apt to be distinctly localized and are called gummata. They sometimes appear as small aneurism-like swellings upon the periphery of the vessels (gummatous periarteritis), but may grow to quite large tumor-like proportions and become broken down in the center. Sometimes, as often at the base of the brain, this exudation may be more diffuse and involve pretty generally the vessels of the meninges of the floor of the cranium, though they are more rarely seen in that over the vertex (gummatous meningitis).

Besides these two very distinctive lesions of syphilis, we commonly have associated therewith a proliferation of the subendothelial lining of the vessels which may seriously encroach upon or even wholly obliterate their lumina. This *obliterative endarteritis* is not peculiar to syphilis, but a very common result of its invasion.

The whole process then is due to the irritating effects of the syphilitic microbe, and the gummata are but secondary products of the inflammation, much as pus is the product of the invasion of the tissues by the ordinary cocci.

Besides these direct and primary lesions of the brain due to syphilis, there are two degenerative diseases chiefly affecting the nerve cells of the brain which are most commonly produced by the remote effects of the syphilitic poison in the blood. They generally come on after the patient has been apparently cured of all syphilitic symptoms and are thought to be produced by the action of ptomaines in the blood rather than to the activity of the microbe itself. They are generally spoken of as the post-syphilitic brain degenerations and include clinically the diseases known as locomotor ataxia and paresis. The scope of this paper, however, will not allow of but a passing reference to these last two important and common brain affections.

SYMPTOMATOLOGY AND COURSE.

There are so many slight variations in the symptoms and their combinations that it is hard to form a mental picture of the disease type merely by mentioning *seriatim* the symptoms that *may* occur. Perhaps it will be better to review in brief a clear case of the affection which came under our care within the last year, and which presented nearly all the classical symptoms in their usual order.

E. W. was born with a silver spoon in his mouth, and early in life was fostered with especial care on account of a neurotic tendency in the family. This spoiling process but tended to increase this natural tendency in the child who, though soon well educated and traveled, became wilful and wayward, and could not deny himself anything that appealed to his appetite or fancy. While a student abroad he formed many *liaisons*, and, 't was whispered, had a wife in England, France and in Germany. Six years ago he met his death by seducing what he supposed to be a vestal virgin from one of the shrines in Japan. From her he contracted syphilis. He took treatment from leading specialists, generally selecting that form proposed that was the easiest. He also took special "postgraduate courses," so to speak, at some of the baths in Germany. All outward

evidences of the affection disappeared, and on the outbreak of the Spanish War he enlisted and went through the hardships of a year's campaign in the Philippines. This evidently acted as an exhausting and hastening factor, for a short time after returning home he was taken with gradually increasing neuralgic pains in his head, especially over the vertex and just above, and in, the eyes. These pains were very persistent and did not react well to treatment. Besides the inability to use his eyes on account of the pain, he found his eyesight rapidly failing, and took to strong glasses, which but partially corrected the defect.

Within a fortnight of the onset of symptoms he grew so bad that his father took him to a sanatorium near Boston. This, however, not proving satisfactory he was removed to a second. While at the latter place, he suddenly had a shock with partial paralysis of the left side and ptosis of the right eyelid. This grew better in a few days and he was sent to the Highland Spring Sanatorium in October, 1901, about one month after the first onset of symptoms.

On entrance he appeared fairly well nourished and presentable, but acted somewhat as if slightly intoxicated. His gait was unsteady, shuffling and at times markedly ataxic. He dragged his left foot more than his right; knee jerks were slightly diminished, ankle clonus not present; the eyes reacted both to light and accommodation, and Romberg's test was negative. There was a marked iodide eruption on the skin, as evidence of recent vigorous treatment. His hand grasp was weaker than normal. He wandered about, taking but little interest in conversation, as if in a brown study, made irrelevant remarks and appeared half asleep. If aroused, however, by vigorous questioning, he would brighten up and show nearly normal intelligence and judgment on nearly any topic, with evidently no serious impairment of memory for the distant past. Of recent events he was not so sure and sometimes entirely at fault. He complained of a nearly constant headache, and realized somewhat the difficulty he experienced in making the finer movements with his hands, as in buttoning his clothes or in writing. He fumbled everything he touched from lack of accurate sensations and coordination rather than from definite tremor of the muscles. His appetite and digestion were good. Occasionally he would suddenly become nauseated and refuse a meal, or vomit what he had eaten without much nausea.

He improved somewhat for a fortnight and then began to show more serious mental defects. He became untidy in his habits, more forgetful and careless, and then so confused as to be definitely deluded. He complained of a peculiar sensation in his head which made him think he was on a steamer at sea, and he would move in his seat on the piazza as if righting himself to the pitch of the vessel and perhaps startle every one present by a question as to how soon would we all reach Liverpool at this rate of speed. Thus a few delusions and hallucinations became perceptible, probably from transitory circulatory disturbances in the brain. They were never very marked and not at all troublesome. His speech all along had grown thick and inaccurate in expression, drawling, with the occasional elision of

several words at the end of a sentence as if he had changed the current of his thoughts and forgot what he was going to say. For days at a time he would be very sleepy and rouse only for food. Again, at other times, he would have abnormal thirst and drink large quantities of water, though it was cool weather. This was likely to be accompanied by marked polyuria.

On Nov. 10 he had another shock which caused complete paralysis and coma. His family physician, brought up in consultation, pronounced it apoplexy and refuted our opinion and said he would not live twenty-four hours, certainly not one week, so complete was the paralysis which involved even the nerves of the forehead. He lay thus for several days, and we had to resort to tube feeding. He gradually grew stronger, although never after was he able to leave his bed except to lie on a reclining chair.

His special attendant had to care for him as for a helpless child, although the paralysis passed away from the right side completely and to a great extent from the left. There still remained on the left more or less spastic rigidity. He developed bilateral bed sores, and in fact every area of pressure soon became broken down and would not heal. He could understand fairly well, but his mind was weak, and gradually he failed to recognize members of his own family, became slowly very much demented, and finally died after an attack of a shock-like character, with depression of the heart, on March 12, 1902, just six months after the onset of symptoms.

At autopsy the brain appeared somewhat softer than normal. The dura and pia were slightly thickened and more opaque over the base and in small patches over the vertex, and the arteries showed some general thickening with sclerotic patches. On section the corpus striatum presented a peculiarly soft, yellowish, degenerated appearance over an area on each side larger than a walnut and corresponding to the area of supply of the middle cerebral artery. Under the microscope the brain tissue in these areas was plainly in process of fatty degeneration with destruction of the medullary sheathes. The walls of the finer vessels were much thickened and the channels in places nearly occluded. Evidently the essential cause of the fatty degeneration was the poor blood supply afforded by the terminals of the middle cerebral artery. No definite gummata were found. The meningitis of the base and vertex probably accounted for the headache and cerebral nerve palsies, while the hemiplegias were probably due to acute congestions or edemas of the brain substance, consequent upon some new occlusion of an arterial branch.

DIAGNOSIS.

The diagnosis of cerebral syphilis often presents very great difficulty from the almost innumerable series of combinations of functional impairment that may be complained of. Indeed, it is not always possible at a first examination, or even on several, to be certain of the localization of the principal lesions on account of the varying blood supply and the rapid changes of the symptom groups. There are no pathognomonic symptoms or group of symp-

toms. Their general course and variety help us just as much as the individual symptoms themselves.

The chief points that help to a suspicion of the disease are:

- (1) Headache and vertigo.
- (2) Nausea and vomiting.
- (3) Optic neuritis.
- (4) Cranial nerve palsies or paralyses.
- (5) Apoplectiform attacks, or more gradual attacks of somnolence or coma, with partial hemiplegia.
- (6) Irritability and general mental failure.
- (7) Polyuria and polydipsia.
- (8) Marked remittent character to all the symptoms and their changeability.

The chief danger lies in overlooking a persistent headache, or other single symptom in an otherwise apparently healthy patient until some serious mishap, as hemiplegia or organic change, like optic neuritis, supervenes. Very many of these cases have come from the best practitioners and even specialists, and have been treated for a long time for some defect in the secretion of hydrochloric acid in the stomach, or lack of motility of the small intestine, which has, they think, caused all the headache and nausea and dizziness. The character and grouping of the symptoms, not their mere presence or absence, is an important point. The usual time of onset is about the third year after primary infection, and because more males than females contract syphilis, nervous syphilis occurs seven times more often in men than in women.

As first observed by Lancereux, definite localized brain syphilis may be noticeable by the appearance of headache and facial paralysis and optic neuritis as early as the first month after the primary chancre. In such rare cases, anti-syphilitic treatment is apt to be more successful and the general prognosis far more favorable than in others where the brain symptoms come on in the late stages of the disease. [Since writing the above a subsequent similar case showed marked facial paralysis on both sides only *four days* after the first appearance of the secondary eruptions and before they had become general over the body. In less than three months under the usual rigorous measures, the paralysis and other associated nervous and skin symptoms had disappeared, except a slight remaining weakness in the muscles of the right cheek.] Many cases in the late periods run a similar course to that of true general paralysis, from which it is hard to distinguish them.

PROGNOSIS.

As before stated, those cases occurring early in the course of the general systemic infection offer usually the best chance for recovery, which may be complete or partial, according to the amount of brain tissue destroyed. If there has been hemiplegia, with much cerebral degeneration, some of the results will ever after remain. If the symptoms are arrested when simply caused by pressure from exudation about a nerve or nerve center, nearly complete recovery may be looked for. In the later cases, in which the lesions include a more chronic arterial change and gradually lessening blood supply to the affected area, the results are less encour-

aging, although still more hopeful than for other forms of intracranial new growths. The fatal cases usually live from six months to three years. The duration depends, of course, considerably on the special location of the principal lesions.

TREATMENT.

As a prophylactic against inducing the disease to gain a more certain foothold on the nervous system, it is wise to warn all recently infected syphilitics to "take life easier" than usual, as part of their medical treatment, to avoid alcoholic and other excesses, and especially worry and mental strain. On account of the undoubted danger of cerebral involvement, even in the early "secondary" stages, it is safest to pursue the mixed treatment almost from the start, and even after all external signs of the disease have disappeared, to have the patient take regular doses of iodide of potassium up to the end of the third year. Indeed, special care should be exercised during this third year, as it is then that the majority of the cerebral cases first define themselves.

When undoubted cerebral symptoms occur, it seems the general consensus of the best opinions that iodide of potassium is our chief mainstay, and that to secure any very definite results very large doses have to be administered, even to the utmost limits of their peculiar tolerance to this drug. No definite maximum dosage can be fixed upon. Some have taken as high as 1,500 grains a day, in divided doses, with apparent benefit. Usually considerably smaller doses will suffice, and marked remission of the symptoms and sometimes very remarkable and startlingly rapid recoveries follow. In other cases, as in the one cited above, no apparent hindrance to the disease process can be obtained, and I have often thought that in these the iodide hastened the degeneration by interference with the assimilation of food. The unsuccessful cases belong especially to those occurring some years after the initial lesion.

Clinical Department.

THE IMPORTANCE OF CAREFUL EXAMINATION AND FREQUENT CULTURES IN DOUBTFUL THROAT CASES.

BY W. P. COUES, M.D., BOSTON,

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THE following case well illustrates the subject of this note: Mr. S. P. D. was seen the evening of Jan. 11, 1902. Three days before he began to feel slight sore throat, not sufficient to keep him from his work. On inquiry it was learned that the patient had seen a little child on Christmas Day who subsequently, about two weeks before I saw my patient, developed diphtheria. Other than this the past history was unimportant.

Examination showed the pharynx uniformly reddened, the tonsils somewhat swollen, and some edema of the uvula; there was no membrane visible. The general condition of the patient was good.

Jan. 12. The appearance of the throat on