lost, where its use prevents the patient receiving the greater chances of cure offered by radical surgical removal of the disease. That this danger is a very real one is shown by the increasing number of easily operable carcinomas, such as epithelioma of the lip and cancer of the breast, which now come to us after a futile and extended trial of the x-ray treatment. This means that in each one of these cases valuable time has been lost, and the chances of permanent cure diminished by the ignorant use of the x-ray. I have no hesitation in saying that the amount of damage for which the x-ray is responsible in delaying and preventing operation in carcinoma cases far outweighs the good it has so far accomplished. In discussing this subject this fact can not be too strongly emphasized.

PERISINUSAL ABSCESS OF THE LATERAL SINUS,

WITH METASTASIS IN LIVER AND STERNO-CLAVICULAR JOINT.*

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Aug. 1, 1901, there came for treatment in the Eye, Ear, Nose and Throat Hospital E. A., a white boy, 14 years of age, suffering with a chronic suppuration of the left ear which began in early childhood. The boy was of healthy parentage, and had been subject to very little illness during his life.

Six years previously he was operated on at the hospital for postnasal adenoids, but did not continue attendance at the clinic for the treatment of his ear, which had been carelessly neglected by his parents. In the latter part of July, a week before returning to the clinic, he had begun to complain of pain in the left side of his head about the ear and in the left temple, accompanied by fever and occasional irregular chills. He was dull and listless, and appeared to be in a state of febrile depression. His mental condition was good, however, the pupils normal, and no manifestations of cerebral disorder were apparent. No evidence of inflammation about the mastoid nor tenderness on pressure. Otoscopic examination revealed a large round perforation in the tympanic membrane and a small quantity of thick fetid pus in the ear canal. No granulations or polypi and no swelling of the canal walls. The pain he complained of was intermittent, deep-seated and at times very intense. Instructions were given to syringe the ear thoroughly three times a day with warm boric acid solution, instill ten drops of a bichlorid-alcohol solution after each cleansing, and to keep the patient under close observation.

He left the clinic, however, and was not heard from until four or five days after, when I was called by the family physician, Dr. Ernst, to see him at his home. His condition then was as follows: Decided typhoid state—dry, brown, coated tongue, sordes on teeth, considerable emaciation and debility. Bowels constipated and some enlargement and tenderness of the liver as elicited by percussion and palpation. Temperature ranging between 100 and 103 F., with slight chills at irregular intervals. Left hemicrania becoming more constant and aggravated, very slight ear discharge. Fearing phlebitis of the lateral sinus and jugular on the side of the affected ear, as an explanation of the pyemic course of the temperature, I made careful search for any sign of inflammation about the mastoid and the course of the jugular in the neck, but could find none. We decided to wait a day longer for the possible appearance of some localizing symptom. When next seen the patient showed no change excepting an increase of the hepatic tenderness. We decided at once to make an exploratory aspiration of this organ, but while Dr. Ernst had gone in

search of his aspiration needle the patient had an evacuation of a large quantity of pus and blood from the bowels. Examination of the stool showed it to be characteristic of liver pus. Evacuations of this nature continued in diminishing quantity during the next few days with some relief to the local pain and the high temperature. The headaches still continued, however, and the asthenic condition of the patient increased. Consent was at last gained to have him removed to the hospital for exploration of the mastoid and sinus for focus of pyemic infection.

Operation.—On his arrival there, August 13, temperature was 102 F., pulse 96 and respiration 28. Being much fatigued by transportation, he was allowed to rest until the following day when, after a free exposure of the mastoid, the antrum was laid open and a small mass of granulation tissue and pus removed. The bone otherwise appeared healthy. The lateral sinus was next exposed in the posterior aspect of the mastoid opening and pus began to ooze from between the sinus wall and the upper part of the wound. This when traced up was found to come from a cavity containing about a drachm of creamy yellow pus in contact with the membranous wall of the sinus. This was freely exposed, evacuated of pus and a gauze drain applied. The sinus seemed to be normal otherwise and exploration brought out a flow of pure liquid blood. There was no evidence, therefore, of thrombophlebitis.

The operation was well endured, but a severe chill came on three hours later and the boy complained of pain about the left sternoclavicular joint. Some swelling and tenderness were discovered at this point. On the following day, August 15, he appeared much brighter, with temperature 99.8, pulse 86, respiration 24; no further complaint of headache. Noticeable redness and swelling at sternoclavicular articulation; also marked tenderness over region of spleen. Hepatic tenderness still persisted and bloody stools continued.

August 16, another chill and rise of temperature to 103 F. Pain in splenic region more intense. August 17, emesis of dark bloody matter. Hepatic and splenic tenderness much diminished. Clavicular joint much inflamed. Mastoid wound dressed and pus cavity thoroughly irrigated. Favorable progress up to August 19, when the occurrence of sharp pains in chest caused me to make a close examination of lungs. Nothing abnormal was found. The clavicular joint became so painful that an incision was made into it, but no pus was evident. August 21, again slight hematemesis. Convalescence thenceforth was uninterrupted and rapid, and in three weeks patient was pronounced completely well.

My chief object in relating the clinical history of this case is to elicit your interest and to evoke some discussion on a subject which, in recent times, has been receiving considerable attention in otologic circles, particularly among our French and German confrères. It is that of septicopyemia of otitic origin in its relation to thrombophlebitis of the lateral sinus.

As time and the occasion do not permit me to enter into a thorough review of such a broad subject in all its phases, I shall limit my remarks to that point on which there has been much discussion, with various theories, and which is still a mooted question in the pathology of one of the gravest complications of middle ear suppurations. That point is the mechanism or the route of infection of the blood current in that class of cases where pyemia develops without apparent involvement of the lateral sinus or the jugular vein. Until very recent years but one form of otopyemia was generally known, characterized by irregular and frequent chills, marked oscillations of temperature and the development of metastatic abscesses, coming on in the course of a purulent otitis media, and which indicated a thrombophlebitis of one of the large venous channels coursing in the vicinity of the temporal bone, usually the sigmoid sinus or the jugular vein.

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Schwartz, in 1867; Dalby, seven years later, and Politzer, in 1882, cited cases, however, in which septico-pyemia occurred as a complication of middle ear suppuration without phlebitic involvement of the lateral sinus or jugular. It is to Körner, however, that credit must be given for having described this form of pyemia as a distinct pathologic entity, though differing but slightly in its clinical aspects from the classic form. Since the publication of this valuable research it is generally accepted that, from a pathogenic point of view at least, there exist two varieties of otopyemia; pyemia of otitic origin with thrombophlebitis, and pyemia of otitic origin without thrombophlebitis.

Recognition of this fact has brought out a number of observations of cases of this latter variety, and hence arose the question of how the infecting germ gained entrance into the circulation for dissemination throughout the body. Among the most recent contributions to the literature on this subject I must call attention to those of Schmiegelow and Laurens, who have both delved profoundly into the study and research along this line and given a thorough exposé of the principal points at issue. I gather from their writings that five different theories have been advanced as to the true pathogenesis of the affection which we shall consider seriatim.

The first is that which has among its strongest advocates Jansen and Leutert, two of our German contemporaries. Both of these excellent authorities ardently contend that the occurrence of pyemia secondary to middle ear suppuration is pathognomonic of lateral sinus or jugular thrombosis. When confronted with the observations of those cases in which liquid blood has been found coursing through these channels they claim that a thrombosis does not necessarily mean an obliteration of the lumen of the vein by a complete clot, but that there exists a non-obliterating or parietal thrombosis where a thin clot forms on the inner wall of the vessel and does not seriously interfere with the circulation, from which the infected embolic clots or the free pus organisms themselves are carried away by the blood current to find lodgement in the lungs, or pass through into the arterial circulation and cause metastasis in remoter parts. This form of thrombosis, it is claimed, occurs more often as a complication of acute than of chronic otitis, as the virulence of the infection gives rise to pyemic symptoms before obliteration of the lumen of the vessel has had time to take place.

This seems an admission on the part of the advocates of this theory in favor of the explanation advanced by Schmiegelow that a parietal thrombosis is simply the beginning of an obliterating thrombosis. To demonstrate either clinically or anatomically in the living subject the presence of an incomplete clot of this kind is extremely difficult and has not, so far as I can ascertain, been yet accomplished. When exploratory puncture of the sinus is made and liquid blood is found to be flowing through it we take it for granted that no thrombosis exists, but we can not say that there may not be a thrombosis limited to the internal coat of the sinus at that point or at some other point above or below it. Hessler and Leutert have both cited cases in which the thrombosis was limited to the bulb of the jugular.

In a case that came under my own observation the sinus was found empty when opened through the mastoid process, but obstructed both above and below by a firm clot. The external wall showed no signs of inflammation at that point. Hessler has found 12 cases recorded where pyemia with metastasis existed and in

which autopsy showed the lateral sinus normal. Mention is not made, however, of the state of the other sinuses and the jugular. On the other hand, Brieger and Schwabach have reported cases of otopyemia of fatal termination in which postmortem examination failed to reveal any affection either of the lateral sinus or the jugular. These facts are, in themselves, proof sufficient that, contrary to the belief of Jansen and Leutert, we may have pyemia of auricular origin without thrombophlebitis of the lateral sinus or jugular.

Another theory is that accredited to Moos, who is of the opinion that where phlebitis of the lateral sinus can not be demonstrated the pyemia is dependent on thrombosis of one or both of the petrosal sinuses. There are no special symptoms to indicate such a condition, and postmortem examination alone can offer the necessary proof.

Hessler propounds a third theory, and is defended by Körner, to the effect that pyemia of this character is due to an osteophlebitis of the small veins in the temporal bone, which become affected in the course of the mastoid inflammation. The phlebitis does not affect the larger blood channels, but the infected emboli are emptied into them and are carried off to find lodgement in distant parts. Such an explanation seems quite plausible and has many adherents, but to my mind does not clearly account for the fact that the metastatic deposits in this particular form of otopyemia are so prone to pass through the lungs and to be disseminated through the arterial system, which is a generally accepted fact. Neither does it find substantiation in the number of cases reported cured by a simple mastoid operation, which, while it demonstrates clearly enough that the diseased mastoid cells may be the origin of the infection, does not indicate the route by which this infection enters the blood current. Fränkel is of the opinion that the lymphatics are the channels of infection, which also merits reasonable consideration, but which, like the other, lacks pathologic demonstration.

Lastly, we have the theory presented by Heyman and Brieger that the pus organisms themselves gain entrance direct into the circulation by penetrating into the osseous capillaries or those of the tympanic mucosa. This accords with the theory of Cohnheim in regard to diapedesis, and is founded on the well-known fact that leucocytes, which, in these suppurative conditions, may be impregnated with the pus organism, can pass through the vessel walls and serve as microscopic germ carriers to disseminate the foci of infection. It is difficult to understand how a body to any extent larger than the leucocyte or the germ itself could pass through the capillaries without being arrested in the lung structure.

On this account more than any other am I an exponent of this last theory, and a close observation of the clinical features and of the pathologic lesions in the case I record tends to strengthen my belief. Here we had to deal with a chronic suppuration of the ear which had invaded the antrum of the mastoid, but otherwise had caused little disease of the bone. The pyemia in this case evidently originated from the collection of pus lying under the dura and in contact with the walls of the lateral sinus.

The external coat of the sinus was scarcely changed by this contact and the lumen was not obliterated by a thrombus. If a parietal clot had formed its presence could not be detected and it must have undergone spontaneous absorption, as the patient made a rapid recovery after the pus cavity had been cleansed, which could

scarcely have happened had a thrombophlebitis been present.

I am of the opinion that either the free germs themselves or the germ-bearing leucocytes burrowed their way through the thin membranous walls of the sinus and gave birth to the metastatic process in the liver and the clavicular joint. An interesting feature of this case not to be left unnoticed was the spontaneous cure of the hepatic abscess by rupture into the bowel.

SYPHILIS IN THE NEGRO.*

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With the advent of his emancipation and his citizenship the negro earned his syphilis and bequeathed it (both by acquisition and heredity) to his posterity, to such a degree that he, in a great measure, became syphilized prior to his becoming civilized. Syphilis obtains in all of its forms in its most classic type in the negro by reason of his habits, his environments, his utter indifference to the disease and his carelessness and delay in having it treated. My experience, covering a period of more than six years in a large clinic composed almost entirely of negroes, justifies me in making the statement that 75 per cent. of that class of negroes who come as patients are syphilitic (either acquired or hereditary). I could present you with a museum of Jonathan Hutchinson's teeth, bridge-shaped noses, rickets, and Parott's disease, showing the result of hereditary syphilis in negro children.

PRECOCITY.

The precocity of syphilis in the negro is striking. I have the opportunity in hospital work to see it in both races. In the negro it is not uncommon to encounter the chancre, the mucous patches, the secondary skin eruptions, the iritis, the general periostitis—especially in the sternum, the tibiae and the costal cartilage—and the high grade of syphilitic fever existing simultaneously: fulminating syphilis or acute syphilis, if you please, overwhelming in nature, producing profound systemic infection from which all the tissues of the body suffer, especially the arteries, the heart, the kidney and the brain. It has been said by some—and I am afraid they are not acquainted with the facts—that syphilis in the negro will run its course uninfluenced by medication without deleterious results. I am not prepared to confirm this statement; syphilis leaves its marks that can be readily detected by the close observer.

THE RELATION OF SYPHILIS AND PHTHISIS.

The prevalence of phthisis and its high mortality and its rapid course in the negro has been commented on by nearly all southern clinicians and teachers. In making a review of the subject I have not seen sufficient reasons given for its appalling mortality. I encounter it daily, but have never seen a negro with phthisis recover, it matters not what comforts he may have, what his sanitary surroundings may be, or what precautions he may take after the diagnosis has been made. Acute pneumonic phthisis is the most frequent type observed. I have often seen it run through all the stages and terminate fatally in six weeks, when the first examination had shown it to exist only in its incipency.

The question might well be asked, why this high mortality and this rapid course of phthisis in the negro? Basing my reasons on my clinical observation, I think a rational conclusion can be reached as to a reason for

this mortality and for this rapid course. In the majority of patients coming into the clinic with phthisis well developed, a clear history of syphilis, either recent or remote, can be found. The damage that syphilis has already wrought in the system by its effects on the blood vessels, the heart, the kidneys and the general nutrition brings about a lowered vitality, a want of resistance on the part of the patient to the invading enemy. The field is rich and ready for the entrance, cultivation and growth of the tubercle bacilli. This pre-existing or coexisting syphilis gives the right of way to this captain of the men of death, tuberculosis. Whenever a patient, whether white or black, contracts tuberculosis, having had a recent active syphilis, I always look for a rapid and fatal termination. Tuberculosis writes its seal thus on this class of patients: "All hope abandoned when I enter here." I have often seen acute syphilis and acute tuberculosis coexisting in the negro. They are the mighty monarchs which are yearly sweeping thousands of ignorant negroes from the face of the earth. Unquestionably mixed infection plays a great rôle in the production and mortality of phthisis.

SYPHILIS OF THE PLEURA.

I often encounter insidious accumulations in the thoracic cavity where there has been no history of a previous pneumonia, pleurisy or nephritis, where, to the casual observer, the physical signs are negative and there is no symptoms that would lead one to suspect any trouble in the chest. These insidious accumulations, whether exudates or transudates, inflammatory or non-inflammatory, are marked characteristics of a recent syphilis. I wish to report the following illustrative case:

CASE 1.—May 20, a male negro, aged 25, came to the clinic. He suffers with extreme pain in the abdomen, has a constant desire to go to stool, has much straining with passage of small quantities of mucus and pus; the abdomen is rigid and retracted. I found a pronounced rectitis as far up the bowel as could be seen by the speculum. To the touch the ulcers were hard and indurated. There was marked periostitis in sternum, tibiae, radii and costal cartilages. His temperature was 102, pulse 110. He had no cough, no dyspnea, but absolute fixation of the right side. There was absence of vocal fremitus on palpation, absolute flatness anteriorly, posteriorly and laterally on percussion. Auscultation revealed absence of all respiratory sounds. Aspiration withdrew the typical straw-colored serofibrinous fluid. He gave no history, in spite of close inquiry, of a previous pneumonia or pleurisy. Two years ago, while working on a levee on the Mississippi River he contracted syphilis and took treatment of no consequence except patent medicines. His health constantly failed. Chills and fever, accompanied by this proctitis, developed. He continued with great effort at his work, but was forced to abandon it, and came to Memphis in January last. There was no ascites, enlargement of the liver, edema or albumin in the urine. Dr. Malone of the surgical staff withdrew nearly a gallon of serofibrinous fluid by aspiration.

A striking feature of these accumulations is their enormous quantity. In May, 1900, I withdrew from a male negro, who had about the same history as the one above mentioned, two gallons and a pint at one sitting, with the best results. This case serves merely to point out the frequency of this condition. I can explain these accumulations and their not producing symptoms and physical signs with reference to the chest, only by assuming the gradual onslaught of syphilis spending its force in a measure on the pleurae and lungs. Have we not more syphilis of the pleurae and lungs than is generally supposed? Could our pathologists not give us more light on this question?

*Annual Address before Chicago Medical Society, 1903.