area the breath sounds became harsh and puerile, and frequently suggested bronchial breathing; otherwise there was no change in the quality or character of the respirations.

All other systems were normal.

Course of the Illness.

During the three months in which the child was in hospital the temperature was of a swinging character, rising nightly, and reaching 100° to 104° F. The pulse and respiratory rates remained consistently rapid throughout the illness. The appetite remained good, and there was a small but steady gain in weight. There was no vomiting except after the attacks of paroxysmal coughing, but in the later weeks the conclusion of the vomiting was followed by syncope. The child complained of abdominal pain, but denied the presence of any pain in the chest. During the last ten days there was profound oedema of the face and legs, followed by abdominal distension with free fluid in the peritoneal cavity. The child died on June 29th.

While in hospital the chest was explored on five occasions and no pus was found. X-ray examination revealed peribronchial thickening extending to the right base, and both apices were suggestive of tuberculous infection. There was no marked enlargement of the heart shadow on the plates or screen, though a print showed the increase of shadow more clearly.

Pathologist's Report.

The pathologist's report, kindly provided by Dr. Sellars, states: "*Heart and pericardium:* Weight 175 grams. Extensive fibro-purulent pericarditis with adhesion and partial obliteration of the sac. Some mediastinitis. No valvular lesions. Heart muscle softer and more friable than normal. The pericardium was distended with pus, which was collected chiefly anteriorly to the heart. *Lungs and pleura:* There was no pleural effusion. Some congestion and patches of bronchopneumonia in the lungs. Adhesions between the pleura and pericardium and diaphragm and lower part of posterior thoracic wall. *Liver:* Weight 670 grams; intensely fatty. *Spleen:* Weight 67 grams, congested; Malpighian bodies. *Kidneys:* Weight 97 and 87 grams. The abdomen contained some free, clear fluid; no enlargement of mesenteric glands.

"Microscopically the liver was intensely fatty with much venous congestion. The kidneys and spleen were congested with no focal lesion. The pericardium showed a fibro-purulent pericarditis with adhesions. The condition must have lasted for some time. Some organisms were found in the pericardial exudation; they resembled pneumococci microscopically, but an attempt to confirm this diagnosis by cultural examination was not successful."

The points of interest in this case were both numerous and instructive. First, the x-ray negatives showed mottling of both lungs, with no increase in the cardiac shadow; the prints, however, showed a definite enlargement of the heart. Secondly, there was a persistent loss of resonance over the centre of the right lower lobe. Thirdly, the clinical picture was such that a diagnosis of empyema was made and the chest was explored, with, of course, fruitless results. Fourthly, there was never any increase, on percussion, of cardiac dullness towards the right. The case well illustrates the extraordinary difficulty of the diagnosis of pyo-pericarditis in the young child, and yet after death illustrates a definite chain of symptoms which should possibly be easily recognizable during life.

NON-PURULENT TENONITIS.

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PRIMARY serous tenonitis is a rare disease, with well defined characters and a tendency to recur. It reveals in a striking way the anatomical connexions of the tissue which forms for the eyeball a socket, the capsule of Tenon. That primary serous tenonitis is a distinct pathological entity is now generally accepted. Berlin's view, given in an early edition of the Graefe-Saemisch *Handbuch*, that the clinical appearances are dependent on orbital cellulitis, is opposed in a later edition by Birch-Hirschfeld, who claims for primary serous tenonitis an etiology, clinical expression, and therapy of its own. A purulent form of the disease is less infrequent. It is almost always secondary to severe pyogenic infection of the interior of the eyeball (panophthalmitis). But when the intraocular infection is less virulent it happens that the exudation in Tenon's space does not become purulent but keeps a plastic character. Only in purulent tenonitis do the contents of the space point and escape into the conjunctival sac.

CASE I.—Primary Serous Tenonitis.

After an interval of eight years, a man, aged 56, had a second attack of serous tenonitis. The attacks were very much alike and were confined on each occasion to the right orbit. A steadily increasing pain over the right frontal and temporal regions, together with a quickly forming and considerable swelling of the

eyelids on the same side, marked the onset of each attack. The pain was of great severity, making sleep impossible and giving the man a look of extreme suffering. Not until the lids were separated by the fingers did the patient observe that he saw double. Then also it was revealed that there was a bulky chemosis. So far the description applies to each attack. But on the first occasion the globe was slightly proptosed and immobile, while on the second occasion no proptosis was observed and only the movements of rotation upwards and downwards were lost, leaving weakened abversion and adversion. Four days later improvement had set in. The oedema of the lids and conjunctiva was less, some of the lost motility was regained, and the severity of the pain had diminished. At the end of a fortnight, except for a slight suffusion of the bulbar conjunctiva, the eye was normal; there was neither oedema nor diplopia. No pathological change was noticed in the fundus during the attacks, and the visual acuity remained full. No determining cause of the attacks was found. The accessory nasal sinuses were normal.

CASES II AND III.—Plastic Tenonitis Secondary to Intra-ocular Infection.

Two patients, with septic lacerated wounds, developed unilateral tenonitis in association with metastatic choroiditis. At the outset the clinical picture was that of panophthalmitis; but neither perforation of the globe nor pointing over Tenon's capsule took place. The affected eyes became blind from severe irido-choroiditis and, a few weeks later, one of them was enucleated on account of pain. The operation was a tedious separation of the adherent parietal and bulbar layers of Tenon's capsule.

According to Motais, Tenon's capsule is part of the fascial system in the orbit, which provides the muscles with their aponeuroses and the globe and lacrimal gland with their fibrous coatings. Around the optic foramen the aponeuroses of the muscles blend with the periorbital and the fibrous sheath of the optic nerve; while, in front, as the four recti muscles diverge from one another their aponeuroses with the intermuscular extensions assume a funnel-like form, in which the eyeball is placed, like a ball in a cup. Covering the outer surfaces of the muscles is a delicate fascial sheet, the fascia superficialis, which fuses with the sclera in front of the insertions. From the fascia superficialis of the internal and external recti muscles arise, at points opposite the posterior pole of the eye, the internal and external check ligaments, which gain attachment to the lacrymal and malar bones respectively. On the bulbar surfaces of the muscles there is a tougher and more elastic fascial sheet, which near the scleral attachments of the muscles is reflected on to the surface of the eyeball. The opposed bulbar and muscular layers are lined with endothelium and bound the cleft-like space of Tenon. Some thin connective tissue bands, in which are vessels and nerves, traverse this space.

By means of the emissary channels, through which the venae vorticosae leave the eye, Tenon's space is brought into relation with an intraocular lymph space, the supra-choroidal space; and this relationship readily explains the occurrence of inflammation in Tenon's capsule secondary to pyogenic infection of the interior of the eyeball. The stay-like action of the internal and external check ligaments on the deep tough layer of the rectal aponeuroses makes this layer an effective support for the globe and, with the orbital fat, opposes the backward pressure of the orbicularis palpebrarum. So that a collection of fluid in Tenon's space will be forced forwards and will escape, through the thin fascia superficialis, under the bulbar conjunctiva to form a uniform swelling, the chemosis. Birch-Hirschfeld likens the formation of the chemosis to a safety-valve escape, which keeps the proptosis slight or may even obviate its occurrence.

The restricted motility of the eye and the severe pain that accompanies an effort to move it are marked features of tenonitis. Just as synovitis tends to immobilize a joint and to make movement painful, so with an inflammation of the capsule of Tenon, the socket of the eyeball. In their etiology inflammation of a synovial membrane and of Tenon's capsule may be alike, for of Birch-Hirschfeld's collected cases of primary serous tenonitis rheumatism was the commonest cause.

Only exceptionally does primary serous tenonitis run a course different from that of Case I. Sometimes the visual acuity is so impaired from the effect of the exudation on the optic nerve that it is necessary to make an incision into the parietal layer of the capsule between the external and inferior recti. But usually the use of hot compresses, diaphoresis, and sodium salicylate are followed by a rapid disappearance of the signs and symptoms of the disease.