ered, all the others died. Some lived several years with anemia and enlarged spleens. Some still living (in 1890) after 12 or 14 years.

The article contains nothing but clinical observations. The reports of the examinations are very incomplete and unsatisfactory. From the data furnished it would be impossible to make an accurate diagnosis on any of the cases to which he refers. The only probable explanation seems to me to be that he has reported cases of secondary anemia associated with enlargement of the spleen due to a variety of causes. There were no post-mortem examinations reported in any of the cases except the partial one in 1879.

In 1890, at the first pediatric congress held at Rome, G. Somma and Fede⁵⁴ read papers on anemia splenica infettiva dei bambini. These papers appeared in the transactions of the society in 1891, and were also published in 1890 in the *Arch. Italiano de Pediatria*.

Somma's paper consists of references to the literature, chiefly Italian, and a description of the clinical manifestations of the disease. This description is the same as Cardarelli's, and I need not repeat it. It will suffice to give his conclusions. His references include Gretsels, Pel, Ebstein. These cases, as I have already pointed out, were not cases of anemia splenica. Somma refers to Di Lorenzo as the first to suggest the term "infectious" ("infettiva") in 1890.

Somma's conclusions are as follows:

(1) Anemia splenica infantile, or pseudoleukemia splenica as it is called by others, is an illness prevalent in infancy, more frequent than generally supposed. Adults are not exempt, but it is probable that in these cases the origin dates back to early childhood.

(2) Ignored or unknown until quite recently, it was brought into the domain of modern pathology by Antonio Cardarelli, Luigi Somma and Francesca Fede.

(3) The disease is characterized by severe progressive anemia; chronic enlargement of the spleen which shows special characters not found in other diseases.

(4) Clinically it has certain pathognomonic and accidental phenomena. The first are always present, the latter vary.

(5) In its evolution there are usually 3 stages: Early stage, acme and stage of cachexia.

(6) There are 3 forms of the disease: *a*, Chronic febrile; *b*, chronic afebrile; *c*, chronic with recurrent fever. Any one of the 3 forms may appear in the beginning of the disease.

(7) The true nature of the disease has not been discovered. He believes it to be due to a pathogenic organism that finds conditions most favorable for its growth in the spleen, whence it finds its way into the circulation and there produces the anatomical and functional changes which make up the clinical picture.

(8) The course is chronic. The duration covers a period of from 8 months to 3 years. There are not lacking cases in which the term is extended to adult life.

(9) The result is fatal in most cases; in some the course is prolonged to adult life; in rare cases the patients recover.

(10) The prognosis should be guarded. The treatment is chiefly symptomatic. The indication for splenectomy is still a matter of discussion on account of its gravity. When performed it should be done early in the first period of the disease.

Somma had observed 12 cases up to 1891. He never found the glands enlarged. He does not agree with Cardarelli that there is an acute febrile form, but believes it to be a chronic febrile. He agrees that there is little or no increase in the number of leucocytes. These statements of Somma's are worthy of notice because of their incorrectness.

(1) In his conclusion *No. Three* he says that the spleen shows "special characters not found in any other diseases." The lesions which have been described in the spleen are those of *chronic hyperplasia*. It is necessary merely to consult any textbook on pathology to learn how varied the conditions are that produce this change in the spleen.

(2) In conclusion *No. Seven* he refers to the infectious origin of the disease and its localization in the spleen. There is no evidence to be found in the splenic lesions, the course of the disease, or in the results obtained by bacteriological examination, to warrant such a statement.

(3) He says in reference to the enlargement of the spleen that, aside from malaria, if one finds a very large spleen, in 99 cases in 100 it will be a case of anemia splenica!

(To be continued.)

Clinical Department.

GANGRENOUS APPENDICITIS; GENERAL PERITONITIS; MULTIPLE ADHESIONS; GANGRENE OF BOWEL WALL; PERFORATION OF BOWEL; PURULENT AND FECAL ABSCESES; OPERATION; DRAINAGE; DEATH; AUTOPSY.

BY CHARLES L. SCUDDER, M.D., BOSTON,

Assistant in Clinical and Operative Surgery, Harvard University, etc.

O. M. P., 28 years of age, unmarried.¹ This woman has always had good health. The catamenial periods have been regular and painless. The last regular period was three weeks ago. Two weeks and a half ago she was seized with acute abdominal pain, which was general throughout the abdomen, but slightly more severe in the epigastrium. The day following the initial symptoms the pain was decidedly less. Since that time, every second or third day there have been periods of pain at times, unprovoked by the ingestion of food. There has been vomiting associated with distension of the abdomen. Yester-

¹ Massachusetts General Hospital Records, vol. xlv, p. 8.

day the pain was much more severe than before the abdominal distension was greater, no fecal dejections followed large enemata of water.

Examination.—Facies that of a woman who had suffered pain for some days, the facies of a mild general sepsis. Abdomen uniformly distended and uniformly rigid. No distinctly defined tumor detected. Both flanks tympanitic. Rectal examination finds both cul-de-sacs boggy and slightly lowered. Uterus movable and not abnormally placed. The general condition is poor. The pulse 135 and of poor quality.

Operation disclosed a most interesting and unusual condition of things. The bowel was everywhere adherent to the anterior abdominal wall and to itself, so that it was with difficulty that the peritoneal space was opened. Between the adherent coils of intestine was found puriform material. The bowel was soft and boggy. Over the surface of the gut in many places were seen dark bluish-black areas the size of the tip of the finger, which were soft like wet blotting-paper; through such a softened area it was extremely easy to push with ever so gentle pressure an opening into the bowel. In the left iliac fossa was an accumulation of fecal and of purulent material. Upon wiping dry this abscess cavity, fecal material was seen to flow from a perforation of the bowel attached to the pelvic brim. The peritoneum over these areas of softening of the bowel had lost its shiny appearance. The bowel was necrotic in these various spots. The wound through the abdominal wall was left open for free drainage. Gauze wicks were placed deep to the fecal fistula.

The woman lived about seven days following the operation. No new symptoms developed, save the appearance of an external fecal fistula and progressing weakness.

This case illustrates an extremely uncommon condition of things within the peritoneal cavity associated with an ordinary gangrenous appendicitis. The dark bluish areas of softening of the bowel wall suggested at once an actinomycotic process. The subacute course of the disease with multiple adhesions of the intestine rather tended to emphasize this view of the etiology. Microscopical inspection, however, failed to demonstrate the presence of the ray fungus. The condition of the bowel wall is accounted for by a maceration, or chemical effect due to soaking in a purulent fluid.

An autopsy was performed March 12, 1901, by Dr. J. H. Wright.

Anatomical diagnosis.—Gangrenous appendicitis; diffuse chronic pericarditis with subphrenic abscess and multiple peritoneal abscesses; perforation of intestine; laparotomy wound; fibrinous pericarditis; general anemia.

The body of a woman, 155½ cm. long, said to be 28 years of age, well developed, but greatly emaciated. In the anterior abdominal wall, between the umbilicus and pubes, in the median line a widely gaping wound 8 cm. long. In the base of this wound blackened coils of intestine are appar-

ent. Subcutaneous fat very small in amount. Muscles small. Anterior margin of the liver even with the costal border in the right mammillary line. Between the entire superior surface of the right lobe of the liver and the diaphragm, as well as beneath the right lobe of the liver, an accumulation of a large amount, perhaps in all 400 cc., of a yellow, grumous fluid, and mushy, yellowish masses of a shreddy material. The coils of intestine everywhere are bound together and to the abdominal wall by connective tissue adhesions. On manipulation of the intestines considerable quantity of a blackish fecal material exudes. The surface of the intestines, in the region adjacent to the wound in the abdominal wall, is generally bluish, discolored, and tears with great ease, so that in removing the abdominal viscera en bloc they rupture in several places. This rupturing follows because of the very extensive intestinal adhesions. The coils of intestines are freed from one another and dissected with difficulty. In separating the coils in a number of places small pockets of thick, creamy pus are disclosed.

No ulcerative process is found anywhere in the mucous membrane of the large or small intestine. At one point in the small intestine a small perforation, probably 6 mm. in diameter, is present, which cannot be accounted for as the result of a tear. The mucous membrane bordering on this perforation shows no extension of an ulcerative process, but is of normal appearance. This opening is in relation with what appears to be a necrotic and inflammatory process on the outside of the intestine, and is regarded as the result of a perforation proceeding from without inward, rather than from within outward. There are extensive connective tissue adhesions in the region of the cecum and appendix, but these are no more extensive than elsewhere. Enclosed between the coils of the small intestine and their mesentery an indefinite cavity is present which presents evidence of containing fecal material. Near this cavity, and probably immediately connected with it, the vermiform appendix is found after some search.

The exact relations of the appendix to this cavity, as well as the relations of the cavity itself, are not sufficiently determined, owing to the extensive adhesions and to the necessity of disturbing the normal relations in the course of dissection. The appendix vermiformis over most of its distal extremity is bluish, softened and necrotic in appearance. On opening it a considerable portion of its walls appear to be absent, the loss of substance being margined by necrotic, shreddy material. Over a small part of its length it is dilated, and in this situation presents two small losses of substance extending quite through the wall. The largest of these is about 6 or 7 mm. in diameter.

The uterus and adnexa are normal, but involved in some fibrous adhesions to the intestines. The bladder is not remarkable, except for some red patches on the mucous membrane. Small fibrous

adhesions exist at the apex of each of the lungs. The lungs are not remarkable externally.

Heart.—Pericardial sac generally adherent over wide areas to the heart by a coarsely granular, yellow, in places finely shaggy, layer. This layer is perhaps one-half mm. thick in places. On separating the pericardial sac from the heart, the surface of the heart generally, as well as the entire surface of the pericardial sac, presents this fine, shaggy or granular appearance. A large part of the surface of the left ventricle is dark red in color and dotted over with grayish nodules less than a pinhead in size, in addition to some adherent shreds. There is no excess of fluid in the pericardial sac. Weight of heart 250 gm. Valves and cavities normal; myocardium pale. Stomach and pancreas not remarkable. No ulcers of duodenum.

Liver.—Substance pale, but not otherwise remarkable. Gall bladder not remarkable.

Spleen.—Firmly bound down by fibrous adhesions. The organ is of normal size. On section the substance is red, not diffuent. Follicles and trabeculae visible.

Kidneys.—Capsules free, leaving a smooth surface showing some remains of fetal lobulation. On section the kidney substance is generally bloodless and pale. No atrophy; markings normal. Combined weight of kidneys, 261 gm. Adrenals and aorta normal.

Head.—Not opened.

BACTERIOLOGICAL REPORT.

Cover-glass examination.—Fibrous exudate of pericardium; various bacteria; a few capsule-bearing pneumococci.

Pus of peritoneal cavity.—Enormous numbers of bacteria, many pus cells.

CULTURES ON BLOOD SERUM.

Liver.—One large, gray-white, mucous-looking colony and a few small, gray-white, round colonies. Water of condensation cloudy. Microscopical examination shows bacillus, mucosus capsulatus and unknown bacteria.

Spleen.—Several gray-white, discrete and confluent, mucous-like small to large colonies. Cloudy water of condensation. Microscopical examination shows bacillus, mucosus capsulatus and unknown bacteria.

Pus subphrenic abscess.—Gray-white, mucous-like scum of colonies. Cloudy water of condensation. Microscopical examination shows bacillus, mucosus capsulatus and unknown bacteria.

PLAGUE IN SAN FRANCISCO.—The fortieth patient died of suspected bubonic plague Aug. 30, 1901. The autopsy showed a large left inguinal bubo with hemorrhage, in which plague bacilli were found. The heart muscle showed fatty degeneration, and there were numerous plaques upon the aorta. One lung was edematous, the other congested. The spleen was enlarged and hemorrhagic; the kidneys small, soft and congested. The mucous membrane of the stomach also showed hemorrhages.—*Philadelphia Medical Journal*.

Medical Progress.

REPORT ON PROGRESS IN OPHTHALMOLOGY.

BY MYLES STANDISH, M.D., AND WM. DUDLEY HALL, M.D., BOSTON.

(Concluded from No. 15, p. 411.)

CASES OF CONGENITAL WORD-BLINDNESS.

NETTLESHIP¹¹ reviews an article published in the *Lancet* of May 26, 1900, by Dr. Hinshelwood, concerning individuals who are unable to learn to read. The patients are described as being boys who were bright and intelligent in other respects. They could remember words that they had heard spoken and equally well recognize faces and pictures that they had already seen. The auditory memory of letters, words and numbers was excellent, but there was scarcely any visual memory for letters. Hinshelwood believes this to be a congenital defect of visual memory, due to organic defect in that part of the brain where such visual impressions are stored and recorded, his opinion being based upon the fact that after death, in those cases in which there has been noticed a loss of reading power, there have been found changes in the left supramarginal convolution and angular gyrus. Nettleship describes 5 of his own cases, and makes note of the fact that 8 out of 9 affected were males. From an educational point of view he considers the condition as one of importance, for, if curable, a methodical instruction in reading while the brain cells are yet capable of development should be instituted. It is likewise well worth considering whether such a congenitally defective child might not have a better chance if taught the letters of the alphabet as in the old method, rather than by methods more modern. On the other hand, if improvement is not to be expected the sooner the parents and teachers know it the better. He believes that the best way to detect congenital word-blindness, and to determine how and to what extent it admits of improvement, is to continue in the old plan of teaching children their letters as early as possible. Slight cases may be somewhat improved, but in the other and severer types it may be necessary to abandon the usual methods and educate on other lines. Although the detection is easy in children of well-educated parents, the opposite obtains in the case of the children of the lower classes, and who form a large per cent. of the attendance at the elementary public schools. This subject, which is now receiving attention from medical men, should likewise interest the tutors of backward children.

THE PATHOGENESIS OF CHOKED DISC.

Merz¹² asserts that the experiments were performed mostly on dogs and rabbits, and the conclusions are that elevated intercranial pressure can by itself cause choked disc, but this pressure must be uninterrupted for a certain time. Periodic or transient elevations of intercranial pressure

¹¹ The Ophthalmic Review, March, 1901.

¹² Archiv. f. Augnhiknde, xli, 325.