Suppurative Pylephlebitis with Multiple Liver Abscesses Complicating Regional Ileitis:^{,*}

Review of Literature-1940-1960

Bernard Lerman, M.D., John H. Garlock, M.D., Henry D. Janowitz, M.D.

From the Surgical and Medical Services, The Mount Sinai Hospital, New York, N. Y.

THE COMBINATION of its unusual rarity, the striking clinical features and the diversified surgical implications constitutes sufficient reason to record the story of a patient with regional enteritis complicated by suppurative pylephlebitis with multiple liver abscesses. As far as can be determined, this is the first reported example of this unusual combination of circumstances.

As a natural sequence, inquiry was then made into the general problem of pylephlebitis especially with regard to the influence of the antibiotic era on this formerly regarded extremely grave complication of intra-abdominal infection. A thorough review of the pertinent literature during the years 1940 to 1960 was therefore undertaken and our findings will constitute a portion of this communication.

Case Report

M. K., a 43-year-old white man was admitted to Mount Sinai Hospital on April 15, 1960 with the chief complaint of fever and shaking chills of three weeks' duration, as well as crampy intermittent abdominal pain of one month's duration. The present illlness really began in 1943. While serving as a Navy Officer in the Pacific Theater, the patient developed frequent loose bowel movements. The stools at that time were positive for amoeba. Accordingly, he was treated with emetine and iodochlorhydroxquin. However, the diarrhea persisted, and in 1948, after a diagnosis of regional ileitis was established, an operation was performed which consisted of an ileotransverse colostomy with exclusion of the involved distal ileum. He improved considerably and was asymptomatic until January 1958 when diarrhea recurred. In September 1959, after obstructive symptoms became evident, steroid therapy was begun. Some improvement followed. In January 1960, the patient was admitted to the Grace-New Haven Hospital complaining of severe crampy abdominal pain and fever. Again there was a favorable response to steroid therapy. However, in March 1960, when severe obstructive symptoms recurred he was operated upon. The surgeon found a large inflammatory mass in the mesentery of the small bowel near the site of the previous ileocolostomy with dilatation of the proximal ileum. A second bypass procedure, distal to the previous ileocolostomy, was performed, in continuity. Postoperatively the patient continued his febrile course and, in addition, developed shaking chills. It is important to stress the fact that there was no response whatever to vigorous antibiotic therapy.

Among the positive laboratory findings at that time were a 25 per cent retention of bromsulphthalein, a serum bilirubin of 1.8 mg.%. However, there was no clinical jaundice.

When the patient was admitted to Mount Sinai Hospital, the temperature was 38.8° C., blood pressure—120/80, pulse 124, weighed 102 pounds. Examination disclosed a markedly undernourished chronically-ill white man. The abdomen was moderately distended and tympanitic, with tenderness, both direct and rebound in both lower quadrants. A large tender slightly moveable mass was palpable in the mid-abdomen. The liver edge, palpable two fingers below the right costal margin, was firm, sharp and nontender.

Laboratory data revealed the following: Admission CBC-Hgb. 11.2 Gm., WBC 8,550; Differential-63 polys, 1 band, 23 lymph., 2 monos., 2 eos. The WBC rose to 19,050 on May 25, 1960 with a differential of 76 polys, 12 bands, 9 lymphs. and 3 monos. and came down to 8,400

^{*} Submitted for publication July 13, 1961.



Fic. 1. Barium enema roentgenogram showing the two sites of ileotransverse colostomy with a long loop of somewhat dilated ileum hanging hammock-like toward the pelvis. The recurrent disease was located near the proximal anastomosis. The remainder of the loop was normal.

on July 8, 1960. Hemoglobin fluctuated from a low of 7.7 to 11.6 Gm. on discharge. During his hospital stay the patient received eight units of whole blood. Blood cultures on four separate occasions were negative. Stool guaiacs were persistently negative, as were urines for bile and urobilinogen. Blood urea nitrogen was 9.0 mg.% on admission, rising to a high of 50 mg.% on June 3, 1960 and falling to 22 mg.% on June 7, 1960. Total bilirubin ranged from 0.8 to 1.0 mg.% and transaminase was 27.

Barium enema roentgenograms revealed the two sites of anastomosis with a hammock-like loop of ileum hanging downwards between them (Fig. 1). At the distal ileotransverse colostomy, there was some irregularity of the wall of the colon, but no definite evidence of disease could be demonstrated in the loop of ileum immediately proximal to this site. At the proximal ileotransverse colostomy, a loop of diseased ileum, measuring about eight inches, was noted. This was part of the hammock-like loop stretching between the two anastomoses. Barium also filled the originally excluded loop of terminal ileum. The patient's course in the hospital was stormy. The temperature was septic in type and varied between 37.2° and 39.4° C. (Fig. 2). The patient continued to have shaking chills, anorexia, nausea, and abdominal cramps. Massive antibiotic and steroid therapy had no effect. At various times during his hospital stay the patient received the following antibiotic therapy: Penicillin, streptomycin, sulfathallidine, chloromycetin, neomycin, vancomycin, and erythromycin. In preparation for operation, he was transfused frequently and treated with intravenous fluids and electrolytes.

Operation was performed on April 22, 1960 (J.H.G.). A long midline incision was made extending both above and below the umbilicus. Many adhesions were encountered agglutinating bowel and omentum to each other and to the parietal peritoneum. All the structures were freed. The mass which was felt preoperatively consisted of adherent loops of small bowel, transverse colon, greater omentum, and a markedly thickened small bowel mesentery. After a tedious dissection, the anatomy was finally clarified. The original proximal transverse colostomy was delineated. There was a small area of recurrent ileitis proximal to the anastomosis. This measured approximately three inches in length. Hanging downwards from this anastomosis was a fairly long loop of dilated ileum which ascended toward the left to the second ileotransverse colostomy located proximal to the splenic flexure. The intervening mesentery of the small bowel was markedly thickened and filled with succulent nodes. The original excluded segment of ileum attached to the cecum measured 15 inches. This was atrophic. The following procedure was carried out: both ileotransverse colostomies were taken down and the resulting openings in the transverse colon closed in two lavers. The ileal opening of the distal ileo-transverse colostomy was also closed. The portion of ileum containing the recurrent ileitis segment which had been attached to the proximal transverse colon was excised with a considerable portion of the attached mesentery. The originally excluded segment of ileum was removed with the cecum. An end-to-end anastomosis was now effected between the end of the remaining ileum and the beginning of the ascending colon, thus restoring the alimentary function of the right and transverse colon. It was estimated that the patient was left with approximately 61/2 feet of normal small bowel.

It is important to note that examination of the liver at this time failed to reveal any abnormal findings. It was slightly enlarged but of normal consistency to palpation. The right lumbar gutter was drained and the wound closed in routine fashion. Postoperatively the swinging septic fever continued. Massive antibiotic and steroid therapy had no clinical effect. Repeat chest roentgenograms finally disclosed fixation of the right diaphragm and for the first time there was noted marked tenderness in the right upper quadrant as well as shock tenderness along the right costal arch anteriorly and laterally. By percussion, the liver had enlarged to four inches below the costal margin. At no time throughout the patient's stay in the hospital was clinical icterus demonstrable.

On June 1, 1960 a second operative procedure was carried out (J.H.G.). Through a long right subcostal incision, the abdomen was opened. The liver, except for its enlargement about five inches below its costal arch, appeared grossly normal and no area of softening could be determined. However, because it was quite evident that an abscess must be present, needle aspiration directed upwards and posteriorly produced greenish white pus. After suitable walling off of the peritoneal cavity, a clamp was inserted through the liver substance which was broken down by digital manipulation and a large abscess cavity entered in the substance of the right lobe. Numerous loculated abscesses in the adjacent substance of the liver were disclosed by breaking down, with the finger, the septa separating these various compartments. It was estimated that the total amount of pus evacuated measured 500 cc. A large iodoform packing was placed into the depth of the liver, in addition to numerous cigarette drains; the right lumbar gutter was also drained. To wall

off the peritoneal cavity, the inferior edge of the parietal peritoneum was sutured to the lower anterior surface of the liver. Culture of the aspirated pus revealed Beta hemolytic streptococcus.

During the succeeding 12 days, the temperature reached a lower level and varied between 36.6° and 38.0° C. However, during the third week the temperature again rose to 39.4° C. and assumed the septic course noted prior to operation. Antibiotic therapy was continued during this time.

On the assumption that the liver contained an undrained focus, a third operative procedure was carried out on July 12, 1960 (J.H.G.). Through a small right paramedian incision, a bulging of the lower edge of the liver was noted. Aspiration revealed yellowish-green pus. The opening was enlarged and a multiloculated abscess cavity disclosed containing 100 cc. of purulent material. This cavity was drained in the identical manner of the first one.

The response to this latter procedure was quite dramatic. Temperature dropped to normal and the patient became asymptomatic; antibiotic and steroid therapy was gradually discontinued. Appetite improved rapidly and he tolerated a low residue diet without difficulty. The patient was finally discharged from the hospital on July 24, 1960 with all wounds healed and returned to his home in Connecticut.

The patient remained well until July 31, 1960 when he developed an acute episode of syncope and was admitted again to the Grace-New Haven

FIGURE 2.



Hospital. There, he was noted to have carpopedal spasm, facial muscle rigidity, and frequent generalized muscular spasms. A neurological consultant reported as follows: patient is rational and alert, his only complaint being the onset of severe headache three days prior to admission followed by a sudden fainting spell. Examination reveals a positive Chvostek sign, bilateral; and deep tendon hyperreflexia. Pupils responded normally to light and accommodation and were equal. Pulse–128; blood pressure–150/110.

A diagnosis of calcium and magnesium deficiency was made and the patient was accordingly treated with replacement therapy with an excellent response. All evidence of tetany disappeared. The blood electrolyte levels turned to normal and he was finally discharged from the hospital on August 16, 1960. He continued to receive intramuscular injections of magnesium for six months after discharge from the hospital.

The patient was seen at a regular follow up examination on March 17, 1961, eight months after his discharge from Mount Sinai Hospital. He had gained 52 pounds and was completely asymptomatic. He was tolerating a regular diet and having two formed bowel movements a day. Physical examination at this time showed the well healed scars of his operative procedures, no evidence of liver enlargement and no palpable masses. He had resumed his full responsibilities as a practicing attorney and was engaging in his usual physical activities.

Discussion

A review of the literature pertinent to the subject under discussion has failed to reveal a similar instance of suppurative pylephlebitis secondary to ileitis. In the large experience with ileitis at Mount Sinai Hospital since 1932, this is the only instance of this sequence.

In analyzing the sequence of events in this patient, it would seem that the portal infection had already developed sometime prior to his admission to Mount Sinai Hospital. The possibility of pylephlebitis was not even considered prior to our first operative procedure, especially in the light of the finding of a markedly thickened small bowel mesentery due to extensive lymphadenitis secondary to both the intrinsic disease in the ileum and the double bypass procedure without bowel exclusion. It was believed that with the excision of the diseased segment of ileum, and the performance of a more physiological intestinal anastomosis, the fever would subside.

When, however, the septic temperature, shaking chills and the severe anorexia persisted, other causes were looked for. It was at about this period that the patient began to develop, for the first time, pain and tenderness in the right upper quadrant, and a gradually enlarging right hepatic lobe. The additional symptom of severe night sweats centered attention on the liver as probable seat of suppuration. Curiously, at no time was there a clinical or laboratory evidence of jaundice nor was there any response to massive antibiotic therapy. Only after adequate surgical drainage of the multiple liver abscesses, did the fever subside and the patient go on to a complete recovery.

From the pathological standpoint the evidence was clear that the origin of the pylephlebitis was the large group of infected broken down lymph nodes in the mesentery of the small bowel.

Another most interesting feature of the clinical history of this patient was the development of an acute magnesium and calcium deficiency soon after his discharge from the hospital and the immediate and dramatic response to replacement therapy. It is quite obvious that the patient was in a state of subclinical deficiency during the latter part of his stay in the hospital and that the acute episode developed soon after discharge.

This patient is an excellent example of the importance of magnesium in the body economy and emphasizes the findings of Barnes and Cope⁴ who stress the importance of magnesium requirements in some patients with abnormal gastro-intestinal function who have been maintained for prolonged periods on intravenous therapy.

Suppurative pylephlebitis as a complication of acute appendicitis was first described by Waller, in 1846. Apparently from the recent literature, appendicitis is still the most common etiological agent. In a review of the literature since 1940, 79 cases of suppurative pylephlebitis were assembled (Table 1). Our present patient represents the 80th case for this period. Of this total, 56 (70%) were caused by appendiceal inflammation. Similar findings were presented by Hoffman et al.,17 Leger and Montete²⁰ and Caroli et al.⁷

Although portal vein suppuration may develop from varying degrees of appendiceal infection, it is most usually associated as a complication of either appendiceal abscess or grangrene with perforation.³³ There are, of course, other intra-abdominal infectious processes which may produce pylephlebitis. This is reported in the literature: diverticulitis,³² ulcerative colitis,^{5, 14} perforated carcinoma of the stomach, acute pancreatitis, perforated duodenal ulcer, pulmonary abscess, empyema with pneumonia, carcinomatosis,17 after hemorrhoidectomy,¹ typhoid fever and pyosalpinx,²⁰ epididymitis,² pancreatic or splenic abscess, omphalitis¹⁸ and schistosomiasis.³¹

Suppurative pylephlebitis irrespective of cause seems to occur more frequently in young adult men.^{18, 20, 28} The pathogenesis and the pathological features of this unusual disease have been extensively discussed by numerous authors but the reader is especially referred to the excellent article by Ochsner and DeBakey.28 Markedly increased morbidity and mortality resulting from multiple liver abscess as opposed to the single abscess has been repeatedly stressed. 17, 28, 32

It is apparent that the most important factor in this regard has been the great difficulty in securing adequate drainage of the multiple abscesses of the liver. This point is emphasized in the experience with the patient herewith reported.

The signs and symptoms are now well established consisting mainly of repeated shaking chills, septic fever and drenching sweats.⁹ The onset of this triad after an

TABLE 1. Suppurative Philebilis-80 Cases 1940-1960

Ref. No.	No. Cases	Primary Focus	Dead	Alive
11	1	app.*	1	
6	4	app.	4	
3	1	app.		1
13	1	app.	1	
34	1	?	1	
35	1	app.		1
1	1	post- hemorrhoidectomy		1
23	1	sm. bowel infarct.	1	
23	1	post subtotal gastrectomy		1
15	1	app.		1
12	1	app.		1
33	21	app.	20	1
31	1	schistosoma mansoni	1	
30	1	mucocoele appendix	1	
29	1	app.		1
32	1	app.	1	
32	5	diverticulitis	5	
32	4	?	3	1
5	1	ulc. colitis		1
8	1	app.		1
17	17	**	14	3
25	1	app.		1
19	1	app.		1
24	1	Rocky Mt. spotted fever		1
7	1	app.		1
16	3	app.	2	1
21	1	app.		1
18	3	app.	2	1
22	1	app.	1	
ተተቸ	1	regional ileitis		1
Total	80		58	22

* App.-refers to all degrees of appendicitis including abscess and/or perforation. ** Primary foci: 10 appendicitis; 1"pulmonary abscess;

perforated cancer, stomach; 1 emplements, r puniotary abscess, 1 acute pancreatitis; 1 perforated duodenal ulcer; 1 carcinomato-sis; 1 ulcerative colitis. *** Case report.

appendectomy immediately should suggest the diagnosis. Anorexia, nausea and vomiting, right upper quadrant pain, right rib tenderness, a gradually enlarging and tender liver and jaundice are among the more prominent and common additional clinical features. A polymorphonuclear leucocytosis and radiological evidence of an elevated and immobile right diaphragm are helpful in settling the diagnosis. Blood cultures are usually negative, although Hoffman¹⁷ reported positive cultures in nine of 13 cases.

There seems to be general agreement that needling of the liver under such cir-

TABLE 2. Suppurative Pylephlebilis Treated with Antibiotics

Ref. No.	No. Cases	Primary Focus	Dead	Alive
3	1	app.*		1
13	1	app.	1	
34	1	?	1	
35	1	app.		1
1	1	post hemorrhoidectomy		1
23	1	sm. bowel infarct.	1	
23	1	post subtotal gastrectomy		1
15	1	app.		1
12	1	app.		1
31	1	schistosoma mansoni	1	
30	1	mucocoele appendix	1	
29	1	app.		1
32	1	app.	1	
32	5	diverticulitis	5	
32	4	?	3	1
5	1	ulc. colitis		1
8	1	app.		1
17	17	*	14	3
25	1	app.		1
19	1	app.		1
24	1	Rocky Mt. spotted fever		1
7	1	app.		1
16	3	app.	2	1
21	1	app.		1
18	3	app.	2	1
22	1	app.	1	
*	1	reg. ileitis		1
Total	54		33	21

Mortality rate 33/54 (61%).

* See Table 1.

TABLE 3. Cases Treated with Antibiotics and Surgery

Ref. No.	No. Cases	Primary Focus	Dead	Alive
1	1	post hemorrhoidectomy		1
23	1	sm. bowel infarct.	1	
23	1	post subtotal gastrectomy		1
15	1	app.*		1
12	1	app.		1
32	1	app.	1	
32	5	diverticulitis	5	
32	4	?	3	1
17	3	**		3
16	3	app.	2	1
18	1	app.		1
*	1	reg. ileitis		1
Total	23		12	11

* See Table 1.

** Primary focus not specified in 17 cases reported.

cumstances should be avoided because of the obvious risk of peritoneal contamination.

The treatment of suppurative pylephlebitis was initially suggested by Wilms,³⁶ in 1909, when he indicated the possibility of ligation of the ileocolic vein at its origin after appendectomy had been performed. This was later modified by Braun, in 1913, who suggested ligation of the vein proximal to its junction with the superior mesenteric vein. Other authors 26, 28 reported successful ligation of the superior mesenteric vein at or below the level of the transverse mesocolon and the third portion of the duodenum without the subsequent development of impairment of bowel integrity. Finally, Neuhof,27 in 1913, suggested portal vein ligation and Colp¹⁰ reported three such cases with three fatalities

The above described methods have now largely been abandoned. Prophylactic ligation of the mesenteric veins, draining specific foci of infection, may have a limited sphere of usefulness in selected cases, especially when obvious thrombi can be identified at the time of the initial operation. However, secondary ligations of the mesenteric veins have apparently been abandoned.

There is no question that since the advent of the antibiotic era, there has been a marked reduction of the incidence of septic pylephlebitis secondary to intraabdominal infection. The older clinical surgeons most certainly will note the marked difference during the past 25 years. It was assumed, therefore, that the antibiotics would also have a markedly beneficial effect once pylephlebitis has developed. A survey, however, of the literature would seem to indicate that this has not been the case. Of a total of 80 patients, reported since 1940, 54 were treated with some form of antibiotic therapy or a combination of antibiotic and anticoagulant therapy (Table 2). Surgical drainage of the liver abscesses was carried out in 23 of these (Table 3). The total mortality rate was found to be 61 per cent, 33 deaths among 54 patients. There apparently has been no significant improvement over the mortality figures reported prior to the antibiotic era. In 1938, Ochsner and DeBakey published a mortality of 72.3 per cent.

Despite these discouraging statistics, there is clear evidence to indicate that the best results will be obtained by a combination of early diagnosis, massive antibiotic therapy and adequate surgical drainage of the infected liver. The use of anticoagulants is most controversial, but it would appear definitely that these are contraindicated whenever there are signs of liver impairment as evidenced by the presence of jaundice.

Conclusions

1. A successfully treated instance of suppurative pylephlebitis with liver abscess secondary to regional ileitis is reported.

2. The diagnosis of suppurative pylephlebitis should be entertained when a patient develops, after an operation for intraabdominal infection, the characteristic triad of shaking chills, septic fever and drenching sweats. Localizing signs in the right upper quadrant may appear later.

3. Appendicitis continues to be the most frequent antecedent intra-abdominal infection-56 of 80 reported cases.

4. The total mortality despite antibiotic therapy and surgical therapy still remains greater than 50 per cent. The incidence, however, of pylephlebitis following intraabdominal infection has decreased markedly during the past 15 years.

5. Attention is called to the importance of magnesium and calcium deficiency in patients with deranged gastro-intestinal function and the immediate response to adequate replacement therapy.

Bibliography

- 1. Adlers, N.: Portal Pyemia with Recovery. Lancet, 1:151, Jan. 29, 1944.
- Bakst, H. J. and H. Jeghers: Pylephlebitis of Extraportal Origin. Am. J. of Med. Science, 193:690, 1937.
- 3. Barb, J. S.: Pylephlebitis: Response to Sulfanilamide. Am. J. Surg., 53:356, 1941.
- 4. Barnes, B. A., O. Cope and E. Gordon: Magnesium Requirements and Deficits. Ann. Surg., 152:518, 1960.
- Brooke, B. N. and G. Slaney: Portal Bacteremia in Uulcerative Colitis. Lancet, 1:1206, 1958.
- Busch, J. and A. H. Spivack: Observations on Acute Appendicits. S. G. & O., 70:241, 1940.
- Caroli, J., A. Paraf and J. Leymarios: Les septicémies portales. Revue Médico-chirurgicale des Maladies du Foie de la Rete et du Pancreas (Paris). 323:1, 1957.
- Clark, C. W., Jr. and P. A. Bunn: Treatment of Pylephlebitis Secondary to Acute Appendicits. N. Y. S. J. Med., 53:3007, 1953.
- 9. Colp, R.: Chills in Acute Appendicitis. Ann. Surg., 85:257, 1927.
- Colp, R.: The Treatment of Pylephlebitis of Appendicular Origin. Surg. Gynec. & Obst., 43:627, 1926.
- 11. Crossland, P. M.: Pylephlebitis Complicating Appendicitis. U. S. Naval Med. Bull., **39**: 398, 1941.
- D'Abreu, F.: Suppurative Pylephlebitis with Recovery. Proc. Royal Soc. of Med., 39: 309, 1946.
- Eitzen, O.: Acute Gangrenous Appendicitis Complicated by Pylephlebitis. Ohio State Med. J., 40:325, 1944.
- Felsen, J. and W. Wolarsky: Suppurative Pylephlebitis with Bacteremia in Chronic Ulcerative Colitis. Ann. Int. Med., 33:211, 1950.
- Gamm, K. E.: Penicillin Therapy in Pylephlebitis. J. A. M. A., 128:1159, 1945.
- Havel, J. and V. Zuna: Eitrige Pylephlebitis Appendikalen Ursprungs. Zentralblatt fur Chirurgie (Leipzig), 83:2275, 1958.
- Hoffman, H. L., P. F. Partington, A. L. De-Sanctis: Pylephlebitis and Liver Abscess. Am. J. Surg., 881:411, 1954.
- Klinefelter, H. F., Jr., W. E. Grose and H. J. Crawford: Pylephlebitis. Johns Hopkins Hosp. Bull., 106:65, 1960.
- 19. Leger, L. and A. Durand: Résultat datant de quatre ans d'une anastomose splénorénale

pour pyléphlébite. Memoires de L'académie de Chirurgie, 80:869, 1954.

- Leger, L. and P. Montête: Pyléphlébites suppurées. J. de Chirurgie (Paris), 732:148, 1957.
- Marson, F. G. and M. J. Meynell: Pylephlebitis and Septicaemia Treated with Aureomycin. Br. Med. J., 12:764, Apr. 4, 1953.
- Matiashina, V. M.: On the Diagnosis of Pylephlebitis Developing as a Result of Acute Appendicitis. Klin. Med. (Mosk.), 41:135, 1960.
- McBee, C. J., W. S. Priest, A. Schimberg: Pylephlebitis and Pyogenic Liver Abscess. Amer. J. Surg., 74:194, 1947.
- Michon, P., P. Giroud, A. Larcan, J. Grosdidier, C. Huriet and M. Stricker: Thrombophlébite de la veine porte. Revue medicale de Nancy, 83:213, 1958.
- 25. Milliken, N. T. and H. B. Stryker, Jr.: Suppurative Pylephlebitis and Multiple Liver Abscesses Following Acute Appendicitis; Report of a Case with Recovery. New Eng. J. Med., 52:244, 1951.
- Nanson, E. M.: Vascular Lesions Producing the "Acute Abdomen"-Pylephlebitis or Portal Pyemia. Surg. Clinics N. America, 40:1241, 1960.
- 27. Neuhof, H.: Experimental Ligation of the Portal Vein; its Application to the Treat-

ment of Suppurative Pylephlebitis. Surg. Gynec. & Obst., 16:481, 1913.

- Ochsner, A., M. DeBakey and S. Murray: Pyogenic Abscess of the Liver. Am. J. Surg., 40:292, 1938.
- Piotet, G.: Un cas de pyléphlébite guéri par l'association des antibiotiques a'un anticoagulant. Helvetica chirurgica Acta, 22: 281, 1955.
- Rudinsky, H., C. Isaacson and J. Gold: Suppurative Pylephlebitis of Portal Vein Following Mucocoele of Appendix. S. African Med. J., 30:514, 1956.
- Seife, M. and J. R. Lisa: Diabetes Mellitus and Pylephlebitic Abscess of the Liver Resulting from Schistosoma Mansoni Infestation. Am. J. Trop. Med., 30:769, 1950.
- 32. Sheldon, C.: Portal Pyaemia, Br. J. Surg., 45:351, 1958.
- Soro, Y.: Pylephlebitis and Liver Abscess Due to Appendicitis. J. Internat. Col. Surg., 115: 464, Sept. 1948.
- Tow, P. M.: Suppurative Pylephlebitis. Br. Med. J., 2:435, 1944.
- Wilensky, A. O.: Pylephlebitis Under Penicillin and Sulfadiazine Therapy. N. Y. S. J. Med., 452:2082, 1945.
- Wilms: Venenunterbindung bei eitriger pfortaderthrombose Nach appendicitis. Zentralblatt fur chirurgie, 36:1041, 1909.